A STUDY OF THYROID DYSFUNCTION IN PATIENTS WITH METABOLIC SYNDROME

Dissertation submitted to

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY

CHENNAI

In partial fulfilment of regulations

For award of the degree of

M.D (GENERAL MEDICINE) BRANCH – I

KILPAUK MEDICAL COLLEGE

CHENNAI 600 010

April 2016.

BONAFIDE CERTIFICATE

"This is to certify that dissertation entitled "**A STUDY OF THROID DYSFUNCTION IN PATIENTS WITH METABOLIC SYNDROME"** is a bonafide work performed by **Dr. S.KARTHIKEYAN,** post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfilment of regulations of the Tamil Nadu Dr. M.G.R Medical University for the award of M.D. Degree Branch I (General Medicine) during the academic period from 2013 to 2016."

PROF. DR.C.HARIHARAN, M.D
GUIDE FOR THE STUDY
CHIEF UNIT – IV
DEPARMENT OF MEDICINE
GOVT. KILPAUK MEDICAL COLLEGE
CHENNAI - 10

PROF. DR.S.USHA LAKSHMI, M.D., FMMC.,
PROFESSOR AND HEAD OF DEPARTMENT
DEPARTMENT OF MEDICINE
GOVT. KILPAUK MEDICAL COLLEGE
CHENNAI - 10

PROF. DR.R.NARAYANABABU.M.D., DCH
THE DEAN
KILPAUK MEDICAL COLLEGE
CHENNAI -10

DECLARATION

` "I declare that the dissertation entitled ***"A STUDY THYROID DYSFUNCTION IN PATIENTS WITH METABOLIC SYNDROME"*** is done by Dr.S.Karthikeyan at Kilpauk Medical College, Chennai from March 2015 to August 2015 under the my guidance and supervision to be submitted to The Tamilnadu Dr M.G.R Medical University towards the partial fulfilment of requirements for the award of M.D DEGREE IN GENERAL MEDICINE BRANCH-I."

Place: Chennai

Place: Chennai
Date

PROF.DR.C.HARIHARAN,M.D
GUIDE FOR THE STUDY
CHIEF UNIT – IV
DEPARMENT OF MEDICINE
GOVT. KILPAUK MEDICAL COLLEGE
CHENNAI - 10

DECLARATION

` "I solemnly declare that the dissertation entitled ***"A STUDY THYROID DYSFUNCTION IN PATIENTS WITH METABOLIC SYNDROME"*** is done by me at Kilpauk Medical College, Chennai from March 2015 to August 2015 under the guidance and supervision of Prof. Dr.C.HARIHARAN, M.D., to be submitted to The Tamilnadu Dr M.G.R Medical University towards the partial fulfilment of requirements for the award of M.D DEGREE IN GENERAL MEDICINE BRANCH-I."

Place: Chennai

Date: (Dr.S.KARTHIKEYAN)

ACKNOWLEDGEMENT

"At the outset, I would like to thank my beloved Dean, Kilpauk Medical College Prof. Dr.R.NARAYANA BABU .M.D., DCH. for his kind permission to conduct the study in Kilpauk Medical College for having permitted me to conduct the study and use the hospital resources in the study.

I would like to thank to Professor and Head, Department of General medicine Prof. Dr.S.USHALAKSHMI., M.D., FMMC, Kilpauk medical college for permitting me to conduct this study.

I express my heartfelt gratitude to my unit chief Prof. Dr.C.HARIHARAN, M.D., for his inspiration, advice and guidance in making this work complete.

I am ever grateful to Dr.Venkatesvarloo M.D.,Dch, Registrar for his constant support and guidance.

I am extremely thankful to Assistant Professors of Medicine Dr.SRIDHARAN.P, M.D., and Dr.MALARVIZHI.P, M.D., for guiding me.

I would like express my gratitude to the co-operation and constructive criticism shown by my fellow post graduates. Finally, thank all my patients for their active co-operation in this study, without which this would not have become reality**."**

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No.2/02/2015 Dt:01/02/2015
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study of thyroid dysfunction in patients with metabolic syndrome" - For Project Work submitted by Dr.S.Karthikeyan, Post Graduate in MD (GM), Govt. Kilpauk. Medical College, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

27/3/15

CHAIRMAN,
Ethical Committee
Govt. Kilpauk Medical College, Chennai

27/3/15

CONTENTS

ABBREVIATIONS

MS	-	Metabolic Syndrome
WC	-	Waist Circumference
SBP	-	Systolic Blood Pressure
DBP	-	Diastolic Blood Pressure
FBS	-	Fasting Blood Sugar
TC	-	Total Cholesterol
HDL-C	-	High Density Lipoprotein Cholesterol
TGL	-	Triglycerides
LDL-C	-	Low Density Lipoprotein Cholesterol
FT4	-	Free Thyroxine
TSH	-	Thyroid Stimulating Hormone
BMI		Body Mass Index

ABSTRACT

INRODUCTION

Metabolic syndrome and hypothyroid are both individually risk factors for coronary heart disease. Relation between them is not established so far conclusively.

AIM OF THE STUDY

To study the prevalence and to find the types of thyroid dysfunction in Metabolic Syndrome and to find the association of Thyroid Dysfunction and Metabolic Syndrome.

MATERIALS AND METHODS

A total of 60 Patients with metabolic syndrome fulfilling IDF criteria were selected the study. Detailed history of medication, and anthropometric measurements were noted in a semi-structured proforma. Blood pressure was recorded in right upper limb in sitting posture. After eight hours of fasting, blood drawn for fasting blood sugar, lipid profile and thyroid assay in a single sitting. Then statistical analysis made using SPSS22 and excel.

RESULTS AND OBSERVATIONS

In this study, thyroid dysfunction prevalence is 18.33% among metabolic syndrome patients. Subclinical Hypothyroidism is 15% prevalent in metabolic syndrome patients and Overt Hypothyroidism is 3.3% prevalent. There is no incidence of either overt or subclinical Hyperthyroidism in our study population. The prevalence of thyroid dysfunction and hypothyroidism in metabolic syndrome patients are higher than the prevalence in normal population, which is 5.9% for thyroid dysfunction and 4.6% for hypothyroidism (0.3% overt and 4.3% sub clinical hypothyroidism). Incidence of metabolic syndrome in significantly higher in women (25.8) then in men (8%) with metabolic syndrome

CONCLUSION

Thyroid dysfunction occurs in 18.33% of metabolic syndrome patients. Prevalence of Subclinical hypothyroidism (15.0%) and Overt Hypothyroidism (3.33%) in metabolic syndrome patients which is higher than that of general population. One sixth of metabolic syndrome patients or every sixth metabolic syndrome had Subclinical Hypothyroidism. Prevalence of thyroid dysfunction is much more common in Females with thyroid dysfunction than male. Exclude the presence of Thyroid dysfunction while managing metabolic syndrome patients.

INTRODUCTION

Each year mortality due to coronary artery disease and cerebrovascular disease are on the rise. Though there are multiple risk factors leading to these terminal illnesses, few of these risk factors appear in groups. Characteristic of this group is presence of central obesity and insulin resistance; they also have high blood pressure, high triglyceride levels and abnormal fasting blood sugar levels.

These groups of risk factors are known as Metabolic Syndrome. There is high risk of developing cerebrovascular disease and cardiovascular events in people who have metabolic syndrome. With the changing lifestyle and food habits, there is a raise in incidence of obesity and Metabolic Syndrome.

Thyroid disease is associated with atherosclerotic cardiovascular disease. This association may be in part be explained by thyroid hormone's regulation of lipid metabolism and its effect on blood pressure.

Thyroid hormones have ubiquitous effects and influence the function of most organs. This hormone appears to serve as a general pacemaker accelerating metabolic process and may be associated with metabolic syndrome.(9)

Both Metabolic syndrome and thyroid dysfunction are associated with increased risk of atherosclerotic heart disease. Little is known about the relationship between metabolic syndrome and thyroid dysfunction. Only a

few small studies have been performed.(10,11) In a cross sectional study in 220 metabolic syndrome patients, it was found that subclinical hypothyroidism was prevalent in 16.4% of metabolic syndrome patients.(10) In another study, it was found that metabolic syndrome was prevalent in thyroid dysfunction patients.(11) There is no information available in literature regarding this association in this part of the country. Therefore, the association of thyroid dysfunction with metabolic syndrome was evaluated in this study.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

1. To study the prevalence of thyroid dysfunction in metabolic syndrome.

2. To find the types of thyroid dysfunction in Metabolic Syndrome.

3. To find the association of Thyroid Dysfunction and Metabolic Syndrome.

Review of Literature

REVIEW OF LITERATURE

THE METABOLIC SYNDROME

"The metabolic syndrome is also known as syndrome X, (1) the insulin resistance syndrome, (12) and the deadly quartet. (13) The constellation of metabolic abnormalities includes insulin resistance, glucose intolerance, central obesity, dyslipidemia, and hypertension, as well documented risk factors for cardiovascular disease". When grouped together, they are associated with increased risk of cardio vascular disease. (14, 15) The concept of metabolic syndrome exists at least 80 years. (16) That was first described in the 1920s by Klein, a Swedish physician, as the clustering of hypertension, hyperglycaemia, and gout.

DEFINING THE METABOLIC SYNDROME

Although Reaven GM already highlighted the concepts of insulin resistance and metabolic syndrome in 1988, it was not until 1998 before the first attempt for an internationally accepted definition was put forward. (17) Since then several expert groups have formulated and adapted definitions.

In an attempt to achieve some agreement on definition, and to provide a tool for clinicians and researchers, a WHO consultation proposed a set of criteria. Subsequently, the National Cholesterol Education Program's Adult Treatment Panel (NCEP: ATP III) and the European Group for the Study of Insulin Resistance (EGIR) have formulated definitions. These definitions

agree on essential components – glucose intolerance, obesity, hypertension and dyslipidaemia – but do differ in the detail and criteria.

WHO 1999(18)

"Diabetes or impaired fasting glycaemia or impaired glucose tolerance or insulin resistance plus two or more of the following:

1. Obesity: Body mass index >30 kg/m2 or waist: hip ratio >0.9 in males or >0.85 in females.

2. Dyslipidemia: triglycerides ≥ 1.7 mmol/L or HDL < 0.9(male) or <1.0(female) mmol/L.

3. Hypertension: Blood pressure ≥ 140/90 mm Hg.

4. Microalbuminuria: albumin excretion ≥ 20 microg/min."

EGIR 1999(19)

"Insulin resistance plus two or more of the following:

1. Central obesity: Waist circumference ≥ 94 cm (male) or ≥ 80 cm (female).

2. Dyslipidemia: Triglycerides > 2.0 mmol/L or HDL cholesterol <1.0 mmol/L.

3. Hypertension: Blood pressure ≥ 140/90 mm Hg and/or medication.

4. Fasting plasma glucose ≥ 6.1 mmol/L."

ATP III 2001(20)

"Three or more of the following:

1. Central Obesity: Waist circumference >102 cm (male) or >88 cm (female).

2. Hypertriglyceridemia: Triglycerides ≥ 1.7 mmol/L.
3. Low HDL cholesterol: <1.0 mmol/L (male) or <1.3 mmol/L (female).
4. Hypertension: Blood pressure ≥ 135/85 mm Hg or medication.
5. Fasting plasma glucose ≥ 6.1 mmol/L."

AHA/NHLBI 2005(21)

"Any three of the five constitute diagnosis of metabolic syndrome.

1. Elevated waist circumference ≥ 102 cm (male) or ≥ 88 cm (female).
2. Elevated TGL ≥ 150 mgs/dl or medication.
3. Reduced HDL cholesterol < 40 in men or < 50 in women.
4. Elevated BP ≥ 130/85 mm Hg or medication.
5. Elevated fasting glucose ≥ 100mgs/dl or medication."

IDF 2005

"For a person to be defined as having the metabolic syndrome they must have:

1. Central obesity – waist circumference ≥ 94 cm for European men and ≥ 80 cm for European women. For South Asians – Waist circumference ≥ 90 for men and ≥ 80 for women.

plus any two of the following four factors:

2. Raised TG level ≥ 150 mgs/dl or any specific treatment.
3. Reduced HDL cholesterol < 40 mg/dl in males and < 50 mg/dl in females.

4. Raised blood pressure ≥ 130/85 mm Hg or medication.
5. Raised fasting glucose ≥ 100 mg/dl or previously diagnosed type 2 diabetes."

"A major issue for the IDF consensus consultation was the fact that criteria used for obesity in Asian and other populations could be different from those used in the west. This issue was supported by International Obesity Task Force. (24) They noted that in urban Asians, the body mass index range of 23-24 has an equivalent risk of type 2 diabetes, hypertension, and dyslipidaemia as a body mass index of 25- 29.9 in white people."

PREVALENCE OF THE METABOLIC SYNDROME

A very consistent finding is that the prevalence of the metabolic syndrome is highly age-dependent and differs with different diagnostic criteria. "Females are more prevalent than male all over the world. The prevalence increased from 7% in aged 20-29 to 44% for those aged 60-69 years. (25) The prevalence of metabolic syndrome in Chennai was 11.2% and 41.1% using EGIR and ATP III criteria respectively (26, 27)."

Over the past two decades, a striking increase in the number of people with the metabolic syndrome worldwide has taken place. This increase is associated with the global epidemic of obesity and diabetes; with the elevated risk not only of diabetes but also of cardiovascular disease from the metabolic syndrome.

PATHOPHYSIOLOGY

"The most accepted underlying hypothesis to describe the pathophysiology of the metabolic syndrome is insulin resistance. A major contributor to the development of insulin resistance is an overabundance of circulating fatty acids. (28) Insulin is important to both antilipolysis and the stimulation of lipoprotein lipase." Of note, the most sensitive pathway of insulin action is the inhibition of lipolysis in adipose tissue. Thus, when the insulin resistance develops, the increased amount of lipolysis of stored triacylglycerol molecules in adipose tissue produces more fatty acids, which could further inhibit antilipolytic effect of insulin, creating additional lipolysis. Upon reaching insulin sensitive tissues, excessive fatty acids create insulin resistance by the added substrate availability and by modifying downstream signalling. "Presumably, these biochemical changes in insulin mediated signalling pathways result in decrease in insulin-mediated glucose transport and metabolism in the metabolic syndrome as well."

OBESITY AND INCREASED WAIST CIRCUMFERENCE

For several definitions of the metabolic syndrome waist circumference is included. With increase in intra-abdominal or visceral adipose tissue, a higher rate of adipose tissue-derived free fatty acids go to the liver through the splanchnic circulation. Whereas increase in abdominal subcutaneous fat would release lipolysis product into the systemic circulation and avoid direct

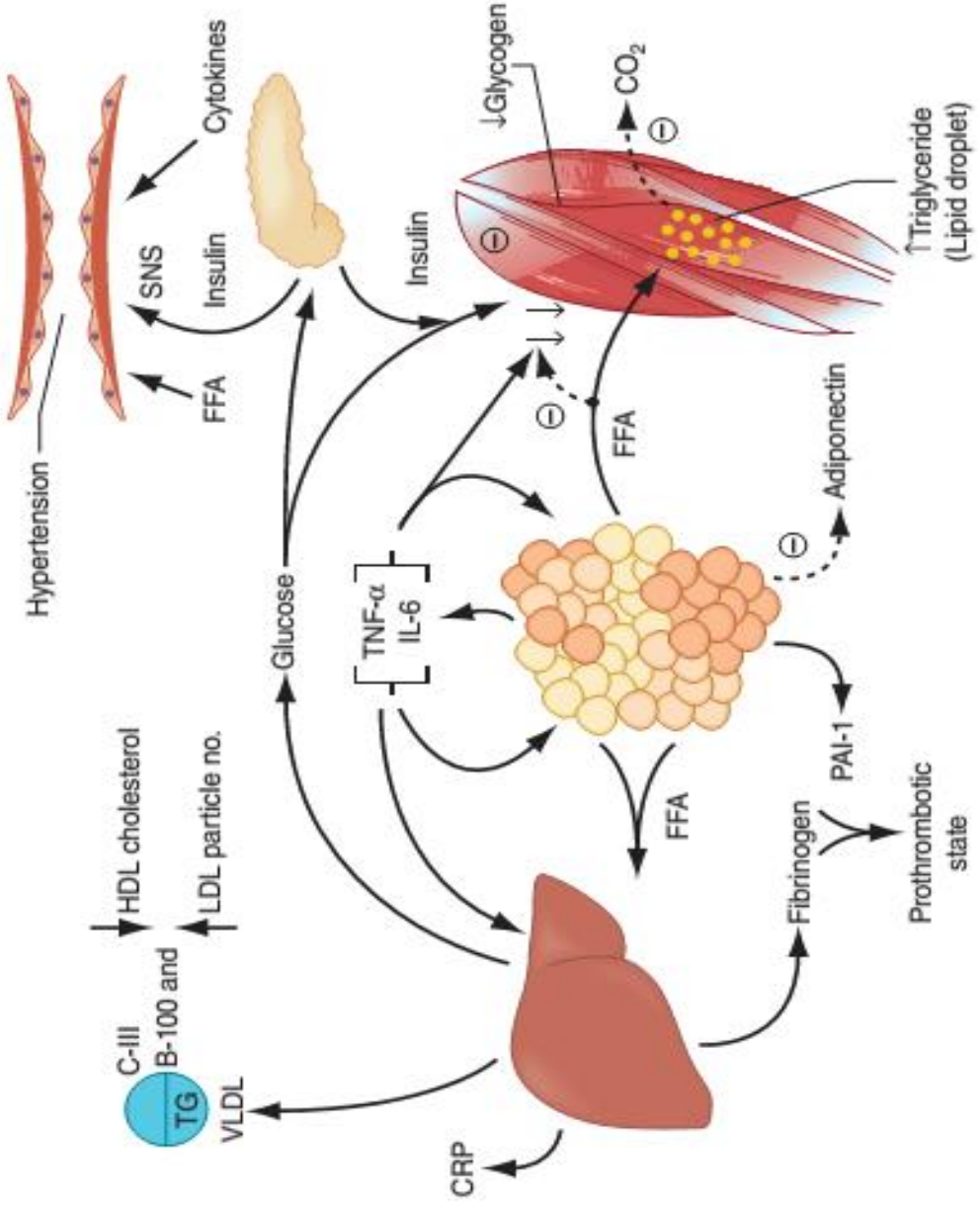


FIGURE 1: **PATHOPHYSIOLOGY OF METABOLIC SYNDROME.** Ref HARRISON'S TEXTBOOK OF INTERNAL MEDICINE

effects on hepatic metabolism. Yet, perhaps by a mechanism related to free fatty acid flux and metabolism, the relative predominance of visceral rather than subcutaneous adipose tissue with increased waist circumference in Asians and Asian Indians renders the relative prevalence of the syndrome higher than African-American men in whom subcutaneous fat predominates.

DYSLIPIDAEMIA

In general, with increase in free fatty acid flux to the liver, increased production of Apo-B containing triglyceride rich very low density lipoproteins occur. In the setting of insulin resistance, increased flux of free fatty acids to the liver increases hepatic triglycerides synthesis; however, under physiologic conditions, insulin inhibits rather than increase the secretion of very low density lipoproteins into the systemic circulation. Hypertrygleridemia is an excellent reflection of the insulin resistant condition and one of the important criteria for diagnosis of the metabolic syndrome. The other major lipoprotein disturbance in the metabolic syndrome is a reduction in HDL cholesterol. This reduction is a consequence of changes in HDL composition and metabolism. In the presence of hypertriglyceridaemia, a decrease in the cholesterol content of HDL results from decreases in the cholesterol ester content of the lipoprotein core with variable increases in triglyceride making the particle small and dense, a function in part of cholesterol ester transfer protein.

This leads to increased clearance of HDL from the circulation. In addition to the clearance of HDL composition of LDL is also modified which is attributable to relative depletion of unesterified cholesterol, esterified cholesterol, and phospholipids with either no change or an increase in LDL triglyceride. “Small dense LDL might be more atherogenic than buoyant LDL because (1) it is more toxic to the endothelium; (2) it is more able to transit through the endothelial basement membrane; (3) it adheres well to glycosaminoglycans; (4) it has increased susceptibility to oxidation; and/or (5) it is more selectively bound to scavenger receptors on monocyte derived macrophages.”

GLUCOSE INTOLERANCE

The defects in insulin action on glucose metabolism include deficiencies in the ability of the hormone to suppress glucose production by the liver and kidney, and to mediate glucose uptake and metabolism in insulin sensitive tissues (i.e., muscle and adipose tissue). Insulin resistance in pancreatic islet-beta cells implies that signals that generate glucose dependent insulin secretion have been adversely modified, and fatty acids are prime candidates. Although free fatty acids can stimulate insulin secretion, increasing and prolonged exposure to excessive concentrations results in fall in insulin secretion.

The mechanism for this alteration has been attributed to lipotoxicity through several potential different mechanisms. In people with genetic

predispositions to development of diabetes, the presumed stress of the insulin resistant environment on beta cell function causes glucose intolerance and ultimately higher risk of diabetes

HYPERTENSION

The relation between insulin resistance and hypertension is well established, and relates to several different mechanisms.

First, it is important to note that insulin is a vasodilator when given intravenously to people of normal weight, with secondary effects on sodium reabsorption in the kidney.

Evidence indicates that sodium reabsorption is increased in white people but not Africans or Asians with the metabolic syndrome. In the setting of insulin resistance, the vasodilatory effect of insulin can be lost, but the renal effect on sodium reabsorption preserved. Fatty acids themselves can mediate relative vasoconstriction. Insulin also increases the activity of the sympathetic nervous system, an effect that might also be preserved in the setting of the insulin resistance.

PROINFLAMMATORY CYTOKINES

The association of the metabolic syndrome with inflammation is well documented. The increases in proinflammatory cytokines including interleukin 6, resistin, and tumor necrosis factor (TNF) and C – reactive protein reflect overproduction by the expanded adipose tissue mass. Evidence suggests that monocyte-derived macrophages reside in adipose

tissue and might be at least in part the source of the generation of proinflammatory cytokines locally and in the systemic circulation. There is increasing evidence that insulin resistance in the liver, muscle, and adipose tissue is not only associated with the abundance of proinflammatory cytokines (and relative deficiency of the anti-inflammatory cytokine adiponectin), but is a direct result of this burden.

ADIPONECTIN

Adiponectin is an anti-inflammatory cytokine that is produced exclusively by adipocytes. Adiponectin both enhances insulin sensitivity and inhibits many steps in the inflammatory process. In the liver, it inhibits both the expression of hepatic gluconeogenic enzymes and the rate of endogenous glucose production. In muscle, it increases glucose transport and enhances fatty acid oxidation, effects that are partly due to the activation of AMP kinase.

In mice decreased circulating concentrations of Adiponectin could be important in producing changes in metabolism consistent with the metabolic syndrome.

CHANGES ASSOCIATED WITH INSULIN RESISTANCE

“LIPOPROTEINS

- Increased apo B
- Decreased apo A-1
- Increased small dense LDL

- Decreased HDL
- Increased Apo C-III
- PROTHOMBOTIC
- Increased Fibrinogen
- Increased plasminogen activator inhibitor 1 (PAI - 1)
- Increase viscosity

INFLAMMATORY MARKERS

- Increased white blood cell count
- Increased Interleukin 6
- Increased tumor necrosis factor alpha
- Increased resistin
- Increased C-reactive protein
- Decreased adiponectin

VASCULAR

- Microalbuminuria
- Increased asymmetric dimethyl arginine

OTHER

- Increased uric acid
- Increased homocysteine
- Non-alcoholic steatohepatitis (NASH)
- Polycystic ovaries syndrome (PCOS)
- Obstructive sleep apnea (OSA).”

MANAGEMENT OF THE METABOLIC SYNDROME

The primary goal of management of the metabolic syndrome is to reduce the risk for clinical atherosclerotic disease. A closely related goal is to decrease the risk for type 2 diabetes in those patients who do not yet manifest clinical diabetes. The first line therapy is to reduce the major risk factors: stop cigarette smoking and reduce LDL-C, blood pressure and glucose levels to recommended goals. Lifestyle modifications are the first line interventions to reduce the metabolic risk factors. The major lifestyle interventions include weight loss, increased physical activity and modification of diet. For individuals at higher risk consideration must be given to specific therapies for the metabolic risk factors. (21) Selective CB1- receptor blockade drugs like Rimonabant significantly reduces the several metabolic risk factors in metabolic syndrome. (29,30)

THERAPEUTIC GOALS AND RECOMMENDATIONS

Abdominal obesity:

Goal: 10% weight loss first year, thereafter continued weight loss or maintain weight.

Recommendation: caloric restriction; regular exercise; behaviour modification

Physical inactivity:

Goal: regular moderate-intensity physical activity.

Recommendation: 30–60 min moderate-intensity exercise daily

Atherogenic diet:

Goals: reduced intakes of saturated fats, Trans-fats and cholesterol.

Recommendations: saturated fat 7% of total calories; reduce trans-fat; Dietary cholesterol < 200 mg daily; total fat 25–35% of total calories.

Cigarette smoking:

Goal and recommendation: complete smoking cessation

LDL-Cholesterol:

"High risk patients are those who have established cardiovascular disease, diabetes, or 10 year risk for coronary heart disease more than 20%. Moderately high risk patients are those with ten year risk of coronary artery disease between 10-20%."

"The cholesterol guideline defined four statin benefit groups

1. all individuals who have clinical atherosclerotic cardiovascular disease (ASCVD), therefore considered "secondary prevention
2. those with LDL cholesterol ≥190 mg/dL without a secondary cause such as a high intake of saturated or trans fats, various drugs, or certain diseases;
3. individuals with diabetes without established cardiovascular disease who are 40–75 years old and have LDL cholesterol of 70–189 mg/dL;
4. those without established ASCVD without diabetes who are 40–75 years old and who have LDL cholesterol of 70–189 mg/dL and a calculated ASCVD risk ≥7.5%."

Goals:

LDL cholesterol < 100 mg/dl (2·6 mmol/L).

“Recommendations:

I. High-risk patients—lifestyle therapies and LDL cholesterol lowering drugs to achieve recommended goal. Moderately high-risk patients—lifestyle therapies; add LDL-cholesterol lowering drug if necessary to achieve recommended goal when baseline LDL cholesterol < 130 mg/dl (3·4 mmol/L).
II. Moderate risk patients (those with 10-year risk of coronary heart disease less than 10%) —lifestyle therapies; add LDL-cholesterol lowering drug if necessary to achieve recommended goal when baseline LDL cholesterol _1·6 g/L (4·1 mmol/L).”

High triglyceride:

Goal: insufficient data to establish goal

HDL-Cholesterol:

Recommendation: High-risk patients—consider adding fibrate (Preferably fenofibrate) or nicotinic acid to LDL-lowering drug therapy

Elevated blood pressure:

Goals:

Blood pressure < 135/85 mm Hg.

For diabetes or chronic kidney disease: Blood pressure < 130/80 mm Hg. Recommendation: lifestyle therapies; add antihypertensive drug(s) when necessary to achieve goals of therapy.

Elevated glucose:

Goal: maintenance or reduction in fasting glucose if _1 g/L (5·5 mmol/L). HbA_{1C} < 7.0% for diabetes.

Recommendation:

Lifestyle therapies; add hypoglycaemic agents as necessary to achieve goal Fasting glucose or HbA_{1C}.

Prothrombotic state:

Goal: reduction of prothrombotic state.

Recommendation:

High-risk patients—initiate low-dose aspirin therapy; consider clopidogrel if aspirin is contraindicated. Moderately high-risk patients—consider low-dose aspirin therapy. Proinflammatory state Recommendations: no specific therapies

THYROID DYSFUNCTION

The thyroid is one of the largest endocrine glands in the body. Thyroid produces three types of hormones, namely thyroxin (T4), triiodothyronine (T3) and calcitonin. Among them $\mathbf{T_3}$ and $\mathbf{T_4}$ are two closely related hormones. Together they play a major role in cell differentiation during

development, and maintain metabolic and homeostasis in adults. Up to 40% of the T4 is converted to T3 by peripheral organs such as the liver, kidney and spleen.

PHYSIOLOGY OF THYROID HORMONES

Thyroid hormone is produced from thyroglobulin (Tg), a large glycoprotein. Inside thyroid follicle thyroglobulin is iodinated on the tyrosine residues. They are coupled via ether linkage. The iodinated thyroglobulin molecules are reuptaked by thyroid follicular cells where they undergo proteolysis and release the newly formed thyroid hormones **T_3 and T_4.**

The first major step in thyroid synthesis is iodine uptake from the gut. iodine absorbed in the gut is converted into iodide and is transported in the blood bound to albumin. It is then actively transferred into the thyroid follicular cells by "Iodide trapping" by sodium iodide symporter. The trapped iodide is oxidized to iodine and combines with tyrosine to form Mono iodotyrosine (MIT) and Diiodotyrosine (DIT) . MIT and DIT are coupled to form T_3 whereas two DIT couple to form **T_4.** Oxidation, Iodination and coupling reactions are catalyzed by "Thyroid Peroxidase". Thyroid hormones thus produced are bound with thyroglobulin until secreted. Once secreted, it is transported in two forms in the blood. One is bound form in which T3 and T4 are bound to plasma proteins namely thyroid binding globulin, pre albumin and albumin. T4 is predominantly

bound to thyroid binding globulin whereas T3 is predominantly bound to albumin. The other form is free T3 and T4. These free forms are in equilibrium with bound form.

In the periphery one third of T4 is converted to T3 by 5' Deiodinase and 45% to rT3 by 5 Deiodinase. They are further metabolized to Diiodothyronine. Only about 13% of T3 is produced from thyroid gland and remaining 87% is formed from T4.

The production of thyroxin is regulated by thyroid-stimulating hormone (TSH), released by anterior pituitary. The thyroid hormones and thyrotropes form a negative feedback loop: TSH production is suppressed when T4 levels are high, and vice versa. The TSH production itself is modulated by thyrotropin-releasing hormone, which is produced by the hypothalamus. The TSH is extremely sensitive to the levels of thyroid

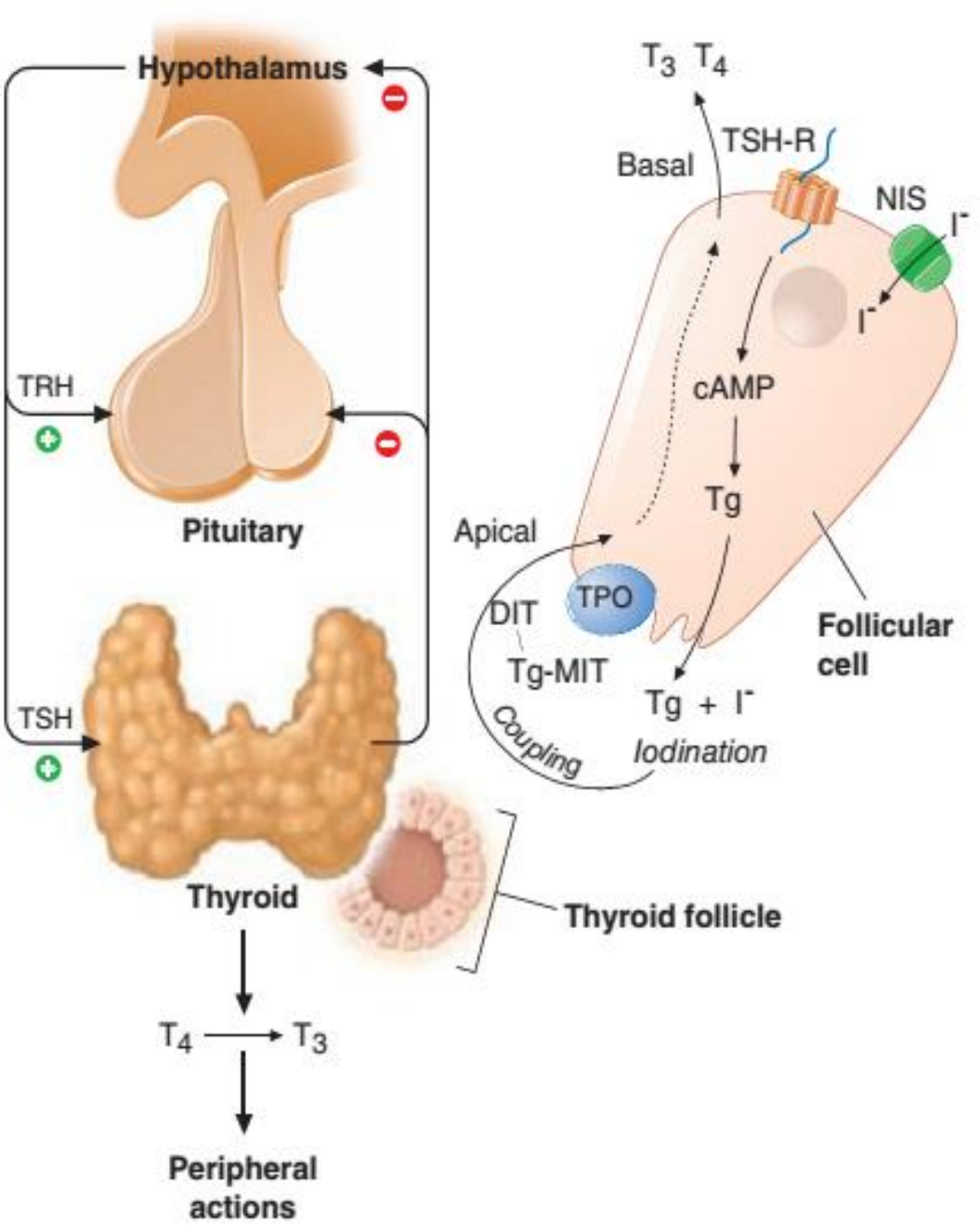


FIGURE 2: **REGULATION OF THYROIS HORMONE SYNTHESIS**

hormones in circulation and can be used as a useful tool in detection of thyroid abnormalities rather than using T4 or T3 levels.

The thyroid dysfunction is simply classified as hypothyroidism, hyperthyroidism, sub clinical hypothyroidism and sub clinical hyperthyroidism depending upon the TSH and thyroid hormone levels.

Clinical status	TSH level	Thyroid hormone
Hypothyroid	High	Low
Hyperthyroid	Low	High
Sub clinical Hypothyroid	High	Normal
Sub clinical Hyperthyroid	Low	Normal

HYPOTHYROIDISM

Hypothyroidism is the condition resulting from lack of the effects of the thyroid hormone on body tissues. Hypothyroidism is a common condition. (31, 32) The overall incidence in the population is approximately 1% to 2 % (33, 34). The serum TSH levels more than 10mU/L and associated with low values of thyroid hormones. Florid hypothyroidism can be diagnosed clinically.

“The symptoms of hypothyroidism in descending order of frequency are:

- Tiredness, Weakness
- Dry Skin
- Feeling Cold
- Hair Loss

- ❖ Difficulty In Concentrating And Poor Memory
- ❖ Constipation
- ❖ Weight Gain With Poor Appetite
- ❖ Dyspnea
- ❖ Hoarse Voice
- ❖ Menorrhagia (Later Amenorrhea)
- ❖ Paraesthesia
- ❖ Impaired Hearing

The signs of hypothyroidism in descending order of frequency are as follows:

- ❖ Tiredness, weakness
- ❖ Dry coarse skin
- ❖ Cool peripheral extremities
- ❖ Puffy face, hands and feet (myxoedema)
- ❖ Diffuse alopecia
- ❖ Bradycardia
- ❖ Peripheral oedema
- ❖ Delayed tendon reflex relaxation
- ❖ Carpal tunnel syndrome
- ❖ Serous cavity effusions.”

HYPERTHYROIDISM

Hyperthyroidism is the condition resulting from the effect of excessive amounts of thyroid hormones in the body tissues. Thyrotoxicosis is a synonym. Grave's disease is the most common cause of hyperthyroidism. Approximately 0.5% to 1% of the population suffers from hyperthyroidism. The TSH levels are suppressed, usually <0.1 mU/L and associated with high levels of thyroid hormones.

"The symptoms of hyperthyroidism in descending order of frequency are as follows:

- Hyperactivity, irritability, dysphoria.
- Heat intolerance and sweating
- Palpitations
- Fatigue and weakness
- Weight loss with increased appetite
- Diarrhoea
- Polyuria
- Oligomenorrhea, loss of libido

The signs of hyperthyroidism in descending order of frequency are follows: Tachycardia; Atrial fibrillation in the elderly

- Tremors
- Goiter
- Warm, moist skin

- ❖ Muscle weakness, proximal myopathy
- ❖ Lid retraction or lid lag
- ❖ Gynaecomastia.”

SUB CLINICAL HYPOTHYROIDISM

According to the latest consensus statement by the American Association of Clinical Endocrinologists, the American Thyroid Association and The Endocrine Society, sub clinical hypothyroidism is defined as an elevated serum TSH level (4.5mU/L to10mU/L) associated with normal total or free T4 and T3 levels. (35) Several alternative names have been proposed to describe this condition and include compensated hypothyroidism, mild thyroid failure, and mild hypothyroidism. The overall prevalence is 2% to 8% in the general population. (33, 34, 36)

SUB CLINICAL HYPERTHYROIDISM

Sub clinical hyperthyroidism is defined as low serum TSH levels (0.1mU/l to 0.4mU/L) associated with normal free T4 and free T3 levels. Sub clinical hyperthyroidism is much less common than sub clinical hypothyroidism. The prevalence is about 2%; it is more common in women, blacks, and the elderly.

NON THYROIDAL ILLNESS

Alteration in serum thyroid hormones occurs in wide variety of illness which predominantly affects the T3 level and no intrinsic disease of thyroid gland is detected. It is variously termed as Low T3 syndrome, Sick euthyroid

syndrome, Non thyroidal illness syndrome and Thyroid hormone adaptation syndrome. This syndrome occurs in wide variety of illness as follows:

a) Acute critical illness and febrile illness such as infections,
b) Myocardial infarction etc.
c) Injuries such as burns, trauma, etc.
d) Surgery
e) Fasting
f) Diabetes mellitus
g) Liver disease
h) Renal disease
i) Ketogenic diet
j) Drugs such as glucocorticoids, dopamine, phenytoin and beta
k) blockers
l) Malignancy
m) Psychiatric illness

In non-thyroidal illness state, initially there is decrease in serum T3 level, both total and free T3 (FT3). This is associated with increase in reverse T3 (rT3).

As illness progresses, there is decrease in serum T4 also, a state called "Low T3, T4 syndrome". Although total T4 level decreases, the free T4 (FT4) remains normal or slightly reduced. In spite of this reduced T3 and T4 level, serum TSH level remains normal or reduced, by which it is

differentiated from primary hypothyroidism. But many studies have showed slight elevation of TSH level in Non thyroidal illness in the absence of hypothyroidism.

THYROID FUNCTION AND THE METABOLIC SYNDROME

It is well documented that hypothyroidism is associated with all the parameters of metabolic syndrome, (32) except increase in fasting blood glucose.

OBESITY

The obesity (increase in waist circumference) is the important symptom and sign of hypothyroidism. More than 60% of hypothyroid patients have obesity (increase in waist circumference). (37) There is decrease in basal metabolic rate and energy metabolism in hypothyroidism.

HYPERTENSION

In hypothyroidism, the hemodynamic alterations cause narrowing of pulse pressure, prolongation of circulation time and decrease in blood flow to the tissues. (38) Systemic vascular resistance is increased in hypothyroidism and results in hypertension. (39) Rotterdam study(6) suggested that there was a twofold increase in risk of atherosclerosis in hypothyroid patients.

LIPID PROFILE

Both the synthesis and degradation of lipid are depressed in hypothyroidism, the latter especially so, the net effect being one of the lipid accumulation, especially of low-density lipoprotein cholesterol and triglycerides. (40) The increase in serum cholesterol in hypothyroidism is accompanied by increased levels of serum phospholipids, serum triglycerides, and the low density lipoprotein cholesterol. The activity of cholesterol ester transfer protein is decreased in hypothyroidism, thus high density lipoprotein cholesterol level reduced in hypothyroidism. (41)

PREVIOUS RELATED STUDIES

Thyroid function is associated with components of the metabolic syndrome in euthyroid subjects. (42) In this population based study there was a negative correlation between thyroid hormone levels (free T4 and free T3) and metabolic syndrome components, Apo B and insulin resistance levels in people with euthyroid state. Free T4 was very significantly related to four of five metabolic syndrome components - waist circumference, fasting glucose, high density cholesterol and triglycerides and insulin resistance level, which assessed by the homeostasis model assessment (HOMA) model. i.e. low normal free T4 was associated with higher triglycerides, lower high density lipoprotein cholesterol, increased fasting glucose and higher waist circumference. Free T3 levels correlated well with systolic blood pressure, triglycerides and Apo-B levels. In insulin resistance

individuals are more susceptible to the association of TSH with higher low density lipoprotein cholesterol and lower high density lipoprotein cholesterol. (43) The morbid obese subjects have higher level of T3, T4 and TSH, probably of the reset of their central thyrostat at higher levels. (44) In a study done by Uzunulu et al., at Japan they have analyzed the prevalence of sub clinical hypothyroidism among 220 metabolic syndrome patients. They found that sub clinical hypothyroidism was 16.4% prevalent in metabolic syndrome patients. (10) One sixth of metabolic syndrome patients had sub clinical hypothyroidism and more prevalent in female gender.

In a study from Nepal, done by Chandra L et al., found that the metabolic syndrome prevalent in 21.1% of thyroid dysfunction patients. (11) They have assessed the association of metabolic syndrome and its components with thyroid dysfunction in 100 female patients. This study found that the prevalence of overall metabolic syndrome was 32%, more in euthyroid group (21/48) than hyperthyroid group (5/24) and hypothyroid group (6/28)

METHODS AND MATERIALS

METHODS AND MATERIALS

Study group	:	Patients with metabolic syndrome attending Medical, diabetic and hypertensive opd.
Study design	:	Single Center Non-randomized cross-sectional study
Place Of Study	:	Govt. Kilpauk Medical College and Hospital
Duration of study	:	6 months
Conflict of interest	:	Nil
Hazards of study	:	Nil

SETTING:

The study was conducted on the out patients attending the Institute of Internal Medicine, Department of Diabetology and Hypertension OPD in Kilpauk Medical College and l Hospital, Chennai.

METHODOLOGY

Detailed history of medication, and anthropometric measurements like height, weight, waist circumference were noted in a semi-structured proforma. Blood pressure was recorded in right upper limb in sitting posture. After eight hours of fasting, blood drawn for fasting blood sugar, lipid profile and thyroid assay in a single sitting.

SELECTION OF STUDY SUBJECTS

The patients who fulfilled the criteria for metabolic syndrome by IDF were taken into the study.

"For a person to be defined as having the metabolic syndrome they must have:

1. Central obesity – waist circumference ≥ 90 cm for men and ≥ 80 cm for women.

Plus any two of the following four factors:

2. Raised TG level ≥ 150 mgs/dl or any specific treatment.
3. Reduced HDL cholesterol < 40 mg/dl in males and < 50 mg/dl in females.
4. Raised blood pressure ≥ 130/85 mm Hg or medication.
5. Raised fasting glucose ≥ 100 mg/dl or previously diagnosed type 2 diabetes"

INCLUSION CRITERIA

The patients who fulfilled the criteria of metabolic syndrome as defined by IDF 2005 were taken up for this study.

EXCLUSION CRITERIA

1. Known Hypothyroid / Sub clinical Hypothyroid
2. Patients with chronic illness.

3. Taking Steroids
4. Severely ill patients
5. Pregnant Women
6. Individuals below 18 Yrs.

CONSENT

Informed consent will be obtained from all participants.

DATA COLLECTION

The data of each patient will be collected on a proforma specially designed for this study and which includes demographic details, past medical history, clinical data and biochemical results will be analysed for statistical significance and correlation.

SAMPLE SIZE

As per formula, sample size was calculated to be 60.

DEFINITIONS

Euthyroidism is defined as

TSH – 0.4 mU/L to 4.5mU/L

FT4 – 0.70 ng/dl to 1.80 ng/dl

Sub-clinical hypothyroidism

TSH – 4.51 mU/L to 10.0 mU/L

FT4 – 0.70 ng/dl to 1.80 ng/dl

Hypothyroidism

TSH – > 10.0 mU/L

FT4 – < 0.70 ng/dl

Sub-clinical Hyperthyroidism

TSH – 0.1 mU/L to 0.4 mU/L

FT4 – 0.70 ng/dl to 1.80 ng/dl

Hyperthyroidism

TSH – < 0.1 mU/L

FT4 – > 1.80 ng/dl

STATISTICAL ANALYSIS

SPSS 12 and Excel were used for data analysis

LIMITATIONS

Small no of study subjects.

FT3 levels not assessed.

CONFLICT OF INTEREST

None

Results and Observations

Results and Observations.

POPULATION CHARACTERISTICS

Total of 60 patients included in the study based on the inclusion and exclusion criteria of metabolic syndrome. Among them 33 were women and 27 were men. Women constitute around 55%of total cases and rest 45% by men.

Age of the women ranges from 33 to 62 years with mean age 45.1 and Standard Deviation 7.6. Age of the men ranges from minimum of 30 to maximum of 67 with mean of 46.5 and Standard deviation of 9.7.

According to age, 7 patients were less than 35 years old. 24 patients were in the age 35-45 age group, 19 were in 45-55 age group and 10 patients were above 55 years. Population characteristics were shown in the Table 1.

Table 1: Descriptive statistics: SEX-MALE

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	25	30	67	46.59	9.775
Valid N (listwise)	25				

a. SEX = Male

Table 2: Descriptive statistics: SEX-FEMALE

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	35	33	62	45.18	7.609
Valid N (listwise)	35				

a. SEX = Female

Table 3: Population Characteristics

AGE GROUP	TOTAL NO	PERCENTAGE	CUMULATIVE PERCENTAGE	Male	Female
Up to 35 yrs.	7	11.7%	11.7%	3	4
36 - 45 yrs.	24	40.0%	51.7%	8	16
46 - 55 yrs.	19	31.7%	83.3%	8	11
Above 55 yrs.	10	16.7%	100.0%	6	4
Total	60	100.0%	100.0%	25	35

Table 4 Frequency table: SEX in study population

	Frequency	Percent	Valid Percent	Cumulative Percent
Male	25	41.67	41.67	41.7
Female	35	58.33	58.33	100.0
Total	60	100.00	100.00	

As we can see most of patients fall in the middle age group from 36 to 55 years, consistent with the changing lifestyle patterns and raising obesity in the middle age group.

Among the sixty study subjects, twenty eight members (47%) fulfilled three parameters for metabolic syndrome, twenty members (33%) fulfilled four parameters and twelve members (20%) fulfilled all criteria for metabolic syndrome.

Table 5: Frequency table: No of criteria positive for MS in subjects

Criteria for metabolic syndrome	Frequency	Percent	Cumulative Percent
3 parameters	28	46.7	46.7
4 parameters	20	33.3	80.0
5 parameters	12	20.0	100.0
Total	60	100.0	

Table 6: Descriptive statistics of the variables in study population

Descriptive Statistics					
	N	Minimum	Maximum	Mean	Standard Deviation
AGE	60	30	67	45.82	8.603
HEIGHT	60	142	180	157.70	8.945
WEIGHT	60	69	106	85.20	8.117
BMI	60	23.96	32.72	26.9952	1.80825
WC	60	85	116	98.08	7.038
SBP	60	100	172	140.10	15.685
DBP	60	66	110	87.23	10.192
FBS	60	96	200	135.85	23.726
TC	60	134	291	194.02	33.090
HDL	60	31	56	44.53	6.342
TG	60	77	307	169.88	52.497
FT4	60	0.17	2.27	1.07	0.265
TSH	60	0.56	154	5.40	19.809
Valid N (listwise)	60				

As waist circumference is absolute criteria to define metabolic syndrome, it is present in all subjects. The following tables show frequency distribution of the other criteria of metabolic syndrome.

Table 7: Frequency table- Diabetes in study subjects

	Frequency	Percent	Cumulative Percent
Absent	2	3.3	3.3
Present	58	96.7	100.0
Total	60	100.0	

Table 8: Frequency table- Hypertension in study subjects

	Frequency	Percent	Cumulative Percent
Absent	15	25.0	25.0
Present	45	75.0	100.0
Total	60	100.0	

Table 9: Frequency table- Triglycerides in study subjects

	Frequency	Percent	Cumulative Percent
Absent	24	40.0	40.0
Present	36	60.0	100.0
Total	60	100.0	

Table 10: Frequency table- HDL in study subjects

	Frequency	Percent	Cumulative Percent
Absent	35	58.3	58.3
Present	25	41.7	100.0
Total	60	100.0	

THYROID FUNCTION TEST RESULTS

The TSH in this study was ranging from 0.56mU/L to 154 mU/L and free T4 levels ranging from 0.17ng/dl to 2.21ng/dl. Patients were grouped into four groups according to the definitions based on TSH and FT4 levels and further statistical analysis was done based on these groups. According to our definitions, 49 patients found to be euthyroid and two patients were hypothyroid. Nine patients had sub clinical hypothyroidism. There were no overt hyperthyroid or sub-clinical hyperthyroidism patients in our study.

Table 11: Distribution thyroid parameters

	N	Minimum	Maximum	Mean	Standard Deviation
FT4	60	0.17	2.27	1.07	0.265
TSH	60	0.56	154	5.4	19.809
Valid N (listwise)	60				

The TSH in this study was ranging from 0.56mU/L to 154 mU/L and free T4 levels ranging from 0.17ng/dl to 2.21ng/dl. Patients were grouped into four groups according to the definitions based on TSH and FT4 levels

Table 12 : Thyroid status of the study population

GROUP	NO	%	MALE	FEMALE
EUTHYROID	49	83.33%	23	26
HYPOTHYROID	2	3.33%	1	1
SUB CLINICAL HYPOT	9	15.00%	1	8
SUBCLINICAL HYPERT	0	0%	0	0
HYPERTHYRODISM	0	0%	0	0

and further statistical analysis was done based on these groups. According to our definitions, 49 patients found to be euthyroid and two patients were hypothyroid. Nine patients had sub clinical hypothyroidism. There were no overt hyperthyroid or sub-clinical hyperthyroidism patients in our study.

According to the age, among patients age less than 35, there were seven subjects. Six were Euthyroid and one is Overt hypothyroid. No subclinical hypo or hyperthyroid in this group.

In the age group 36-45 there were 24 subjects, among them eighteen were Euthyroid and remaining six were Subclinical hypothyroid. There

were no overt hypo or hyperthyroid in this group

Table - 13. Age Wise Thyroid Dysfunction

AGE	TOTAL NO	EUTHYROID	HYPOTHYROID	SUBCLINICAL HYPOTHYROID	SUBCLINICAL HYPERTHYROID
<35	7	6	1	0	0
36-45	24	18	0	6	0
46-55	19	16	1	2	0
>55	10	9	0	1	0

In the age group 46-55, there were 19 members. Among them sixteen were Euthyroid; one is overt hypothyroid and other two were Subclinical hypothyroid. There was no subclinical hyperthyroid in this group.

In the subjects more than 55 years, there were ten members. Among them nine were Euthyroid and remaining one is Subclinical Hypothyroid. There were no overt hypothyroid or subclinical hyperthyroid in this group.

Based on the metabolic syndrome criteria, of those twenty eight patients who fulfilled three of the five risk factors three had thyroid

dysfunction (2-hypothyroid and 1-subclinical hyperthyroid); of the twenty patients who had four risk factors three had thyroid dysfunction (all subclinical hypothyroid); of the twelve patients who had all five risk factors five had thyroid dysfunction (one overt hypothyroid and four subclinical hypo thyroid).

Table 14: Metabolic Syndrome Parameters Wise Thyroid Dysfunction

MS CRITERIA FULFILLED	TOTAL NO	EUTHYROID	HYPOTHYROID	SUBCLINICAL HYPOTHYROID	SUBCLINICAL HYPERTHYROID
3	28	25	1	2	0
4	20	17	0	3	0
5	12	7	1	4	0
TOTAL	60	49	2	9	0

(P valve = 0.36 not significant)

Presence of thyroid dysfunction based on number of criteria present is statistically significant in our study possibly due to limited number of study subjects.

First thyroid status is analysed with respect to distribution among sex.

Table 15: Crosstab Thyroid Status With Respect Sex Distribution

Thyroid status		SEX		Total
		Male	Female	
Euthyroid	Count	23	26	49
	% within SEX	92.0%	74.3%	81.7%
Subclinical Hypothyroid	Count	1	8	9
	% within SEX	4.0%	22.9%	15.0%
Hypothyroid	Count	1	1	2
	% within SEX	4.0%	2.9%	3.3%
Total	Count	25	35	60
	% within SEX	100.0%	100.0%	100.0%

Women has higher incidence of thyroid dysfunction when compared to men with metabolic syndrome.

Table 16: Chi-Square Tests – thyroid status vs sex

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.914[a]	2	.086
Likelihood Ratio	5.617	2	.060
Linear-by-Linear Association	2.275	1	.131
N of Valid Cases	60		
a. 4 cells (66.7%) have expected count less than 5. The minimum expected count is .90.			

Chi-square tests did not any significance of thyroid status distribution with respect to sex in study subjects possibly due to limited number of study subjects.

Analysis of study subjects with respect to number of criteria fulfilled for Metabolic syndrome against sex distribution is as follows.

Table 17: Distribution Number of MS parameter with respect to sex

MS parameter		SEX		Total
		Male	Female	
3	Count	12	16	28
	% within SEX	48.00%	45.71%	46.70%
4	Count	7	13	20
	% within SEX	28.00%	37.14%	33.30%
5	Count	6	6	12
	% within SEX	24.00%	17.14%	20.00%
Total	Count	25	35	60
	% within SEX	100.00%	100.00%	100.00%

Table 18: Chi-Square test – MS parameter vs Sex

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.346[a]	2	.841
Likelihood Ratio	.347	2	.841
Linear-by-Linear Association	.004	1	.947
N of Valid Cases	60		

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 5.40.

Distribution of MS parameter with respect to sex in not significant in our study.

Analysis of thyroid status of the study subjects against number of criteria fulfilled for Metabolic Syndrome by the subjects.

Table 19: Distribution- Thyroid status with respect to no. of MS parameter

Thyroid status		MS parameter			Total
		3	4	5	
Euthyroid	Count	25	17	7	49
	% within MS parameter	89.3%	85.0%	58.3%	81.7%
Subclinical Hypothyroid	Count	2	3	4	9
	% within MS parameter	7.1%	15.0%	33.3%	15.0%
Hypothyroid	Count	1	0	1	2
	% within MS parameter	3.6%	0.0%	8.3%	3.3%
Total	Count	28	20	12	60
	% within MS parameter	100.0%	100.0%	100.0%	100.0%

Table 20: Chi-square test- thyroid status vs MS parameter

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	6.439[a]	4	.169
Likelihood Ratio	6.500	4	.165
Linear-by-Linear Association	3.479	1	.062
N of Valid Cases	60		

a. 6 cells (66.7%) have expected count less than 5. The minimum expected count is .40.

Association between thyroid status and number of criteria positive for metabolic syndrome is not significant in our study

Now finally correlation of the individual parameters of metabolic syndrome in thyroid dysfunction and euthyroid in metabolic syndrome is studied.

Table 21: Frequency table- thyroid dysfunction and euthyroid in metabolic syndrome in study subjects

	Frequency	Percent	Cumulative Percent
Euthyroid	49	81.7	81.7
Thyroid Dysfunction	11	18.3	100
Total	60	100	

Table 22: Distribution of MS parameters in Euthyroid and Thyroid dysfunction

	Thyroid status	N	Mean	Std. Deviation	Std. Error Mean
WC	Euthyroid	49	98.2	6.7	0.961
	Thyroid Dysfunction	11	97.6	8.7	2.609
SBP	Euthyroid	49	140.7	15.7	2.246
	Thyroid Dysfunction	11	137.6	16	4.83
DBP	Euthyroid	49	87.2	10.5	1.5
	Thyroid Dysfunction	11	87.5	9.1	2.751
FBS	Euthyroid	49	138	25.4	3.622
	Thyroid Dysfunction	11	126.1	10.3	3.111
TC	Euthyroid	49	193.3	34.5	4.934
	Thyroid Dysfunction	11	197.4	26.8	8.086
HDL	Euthyroid	49	43.9	6.3	0.902
	Thyroid Dysfunction	11	47.3	6	1.799
TG	Euthyroid	49	171.8	54.7	7.808
	Thyroid Dysfunction	11	161.5	42.8	12.893

Table 23: Independent sample test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
WC	Equal variances assumed	0.562	0.456	0.231	58	0.818	0.547	2.367	-4.191	5.286
	Equal variances not assumed			0.197	12.848	0.847	0.547	2.78	-5.466	6.56
SBP	Equal variances assumed	0.007	0.932	0.573	58	0.569	3.017	5.263	-7.519	13.552
	Equal variances not assumed			0.566	14.65	0.58	3.017	5.327	-8.361	14.395
DBP	Equal variances assumed	0.23	0.633	-0.079	58	0.937	-0.271	3.429	-7.136	6.594
	Equal variances not assumed			-0.086	16.528	0.932	-0.271	3.134	-6.897	6.356
FBS	Equal variances assumed	11.391	0.001	1.527	58	0.132	11.95	7.828	-3.72	27.62
	Equal variances not assumed			2.502	40.124	0.017	11.95	4.775	2.3	21.6
HDL	Equal variances assumed	0.006	0.938	-1.606	58	0.114	-3.354	2.088	-7.534	0.825
	Equal variances not assumed			-1.666	15.46	0.116	-3.354	2.013	-7.634	0.925
TG	Equal variances assumed	0.493	0.486	0.58	58	0.564	10.21	17.615	-25.05	45.469
	not assumed			0.677	18.171	0.507	10.21	15.073	-21.436	41.855

Due to small number of study subjects correlation of metabolic syndrome parameters between euthyroid and thyroid dysfunction is not significant.

Lastly correlation between TSH and FT4 is analysed against metabolic syndrome parameters in euthyroid and thyroid dysfunction is analysed. Correlation is not significant in our study.

Table 24: correlation between TSH and FT4 and MS parameters in Euthyroid

			FT4	TSH
Spearman's rho	WC	Correlation Coefficient	.139	.049
		Sig. (2-tailed)	.341	.740
		N	49	49
	SBP	Correlation Coefficient	.015	.106
		Sig. (2-tailed)	.921	.467
		N	49	49
	DBP	Correlation Coefficient	.067	.028
		Sig. (2-tailed)	.650	.848
		N	49	49
	FBS	Correlation Coefficient	-.080	-.082
		Sig. (2-tailed)	.584	.574
		N	49	49
	TC	Correlation Coefficient	-.066	.103
		Sig. (2-tailed)	.654	.481
		N	49	49
	HDL	Correlation Coefficient	-.007	-.013
		Sig. (2-tailed)	.960	.928
		N	49	49
	TG	Correlation Coefficient	.146	.003
		Sig. (2-tailed)	.317	.985
		N	49	49

Table 25: correlation between TSH and FT4 and MS parameters in Thyroid dysfunction.

			FT4	TSH
Spearman's rho	WC	Correlation Coefficient	.150	-.044
		Sig. (2-tailed)	.659	.897
		N	11	11
	SBP	Correlation Coefficient	.400	-.331
		Sig. (2-tailed)	.223	.320
		N	11	11
	DBP	Correlation Coefficient	.253	-.080
		Sig. (2-tailed)	.452	.814
		N	11	11
	FBS	Correlation Coefficient	-.200	-.014
		Sig. (2-tailed)	.555	.967
		N	11	11
	TC	Correlation Coefficient	-.301	-.096
		Sig. (2-tailed)	.369	.779
		N	11	11
	HDL	Correlation Coefficient	.202	.125
		Sig. (2-tailed)	.551	.714
		N	11	11
	TG	Correlation Coefficient	.400	-.140
		Sig. (2-tailed)	.223	.682
		N	11	11

TABLE 26: DISTRIBUTION OF INDICES

	EUTHYROID		THYROID DYSFUNCTION	
	MEAN	SD	MEAN	SD
AGE	46.35	8.82	43.45	7.49
HEIGHT	158.49	9.02	154.18	8.05
WEIGHT	85.67	7.78	83.09	9.62
BMI	27.01	1.59	26.94	2.68
WC	98.18	6.73	97.64	8.65
SBP	140.65	15.72	137.64	16.02
DBP	87.18	10.5	87.45	9.13
FBS	138.04	25.36	126.09	10.32
TC	193.27	34.54	197.36	26.82
HDL	43.55	6.24	47.27	5.97
TGL	171.76	54.65	161.55	42.76
FT4	1.13	0.24	0.82	0.24
TSH	1.75	0.79	21.6	44.25

(P valve > 0.05 not significant at 5% level)

As there were small no of patients with very high variants, statistically Significant results were not found in our study.

Figure 3: Sex distribution of study subjects

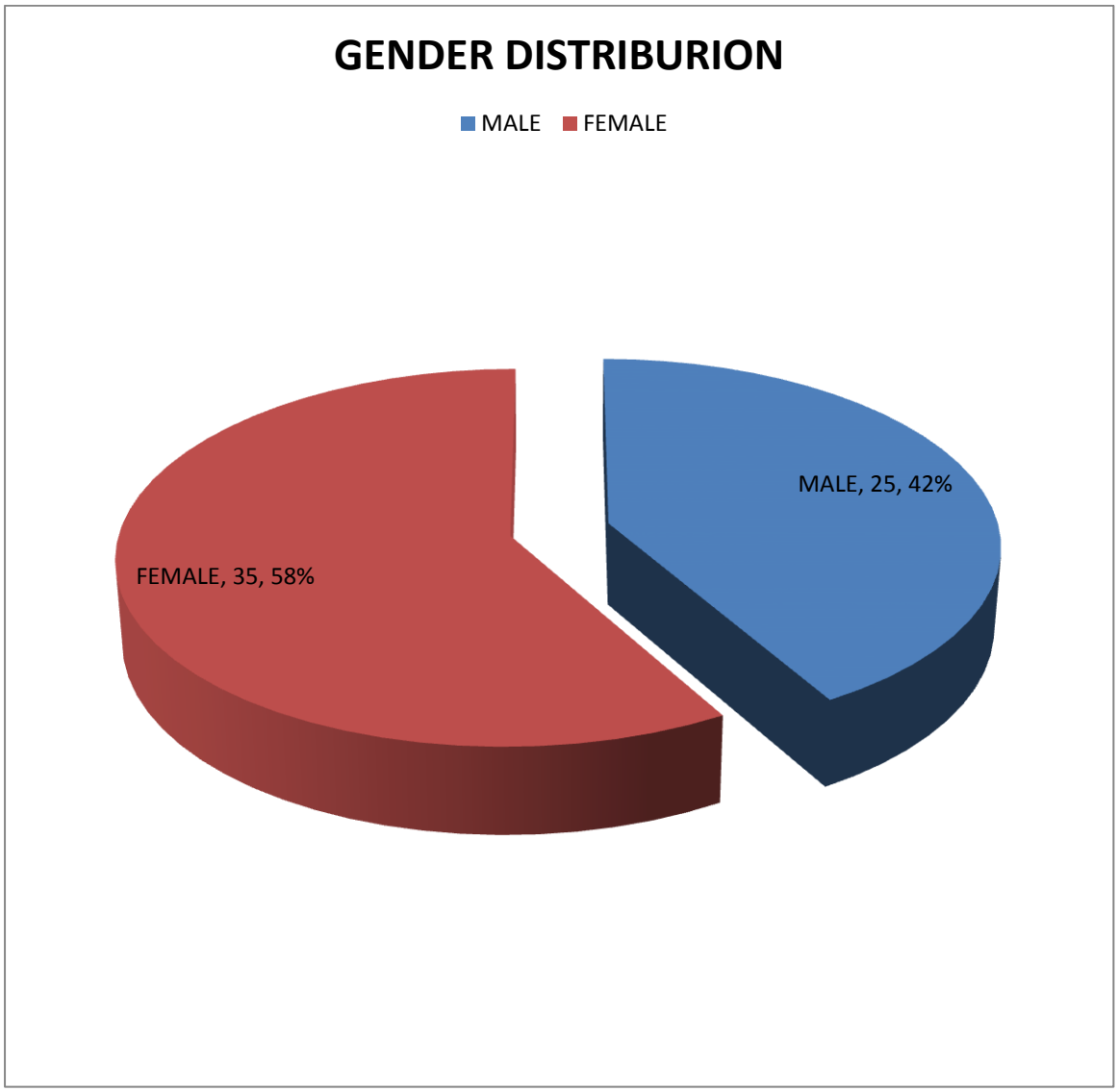


Figure 4: MS parameter distribution

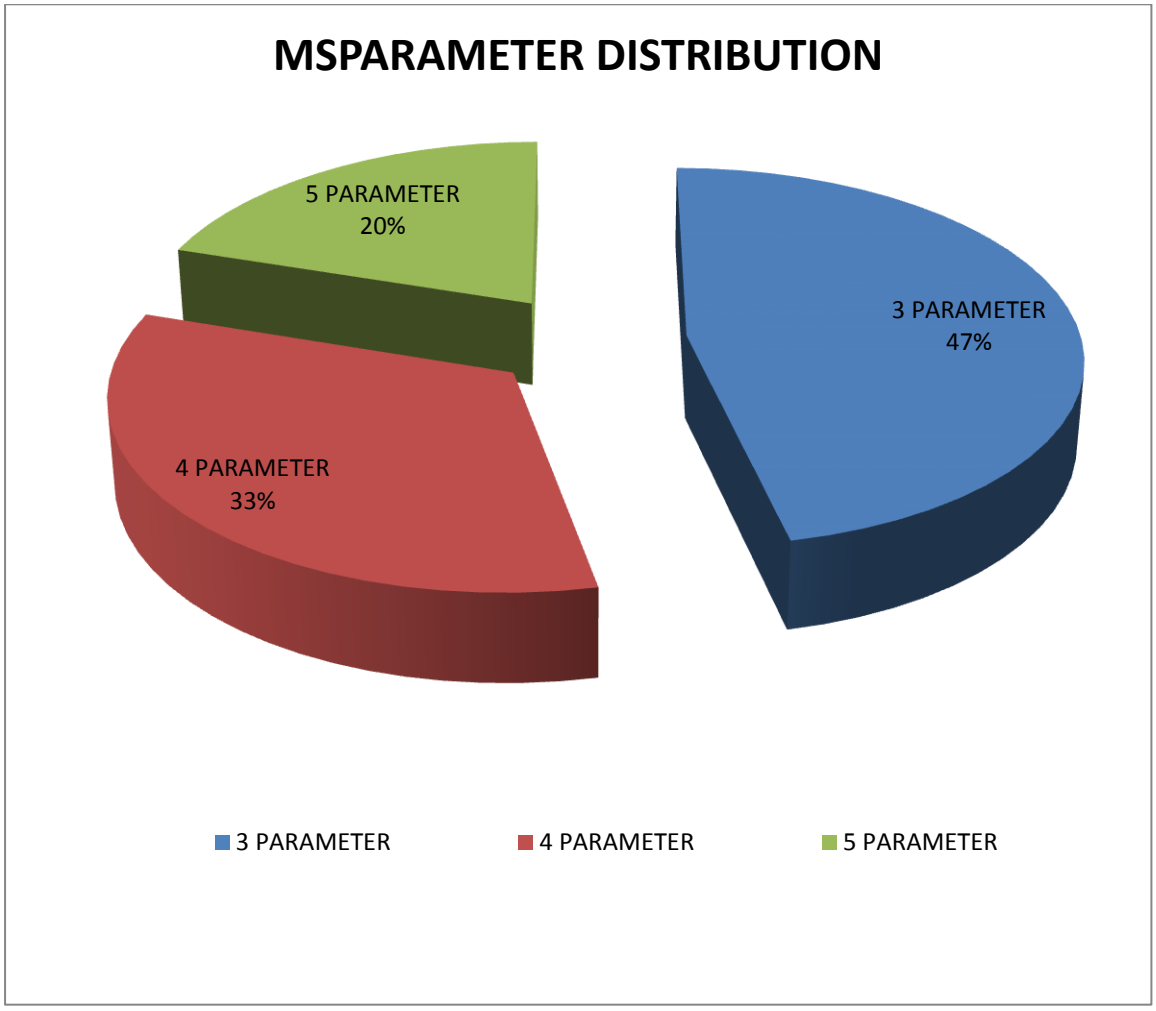


Figure 5: Age distribution of study subjects

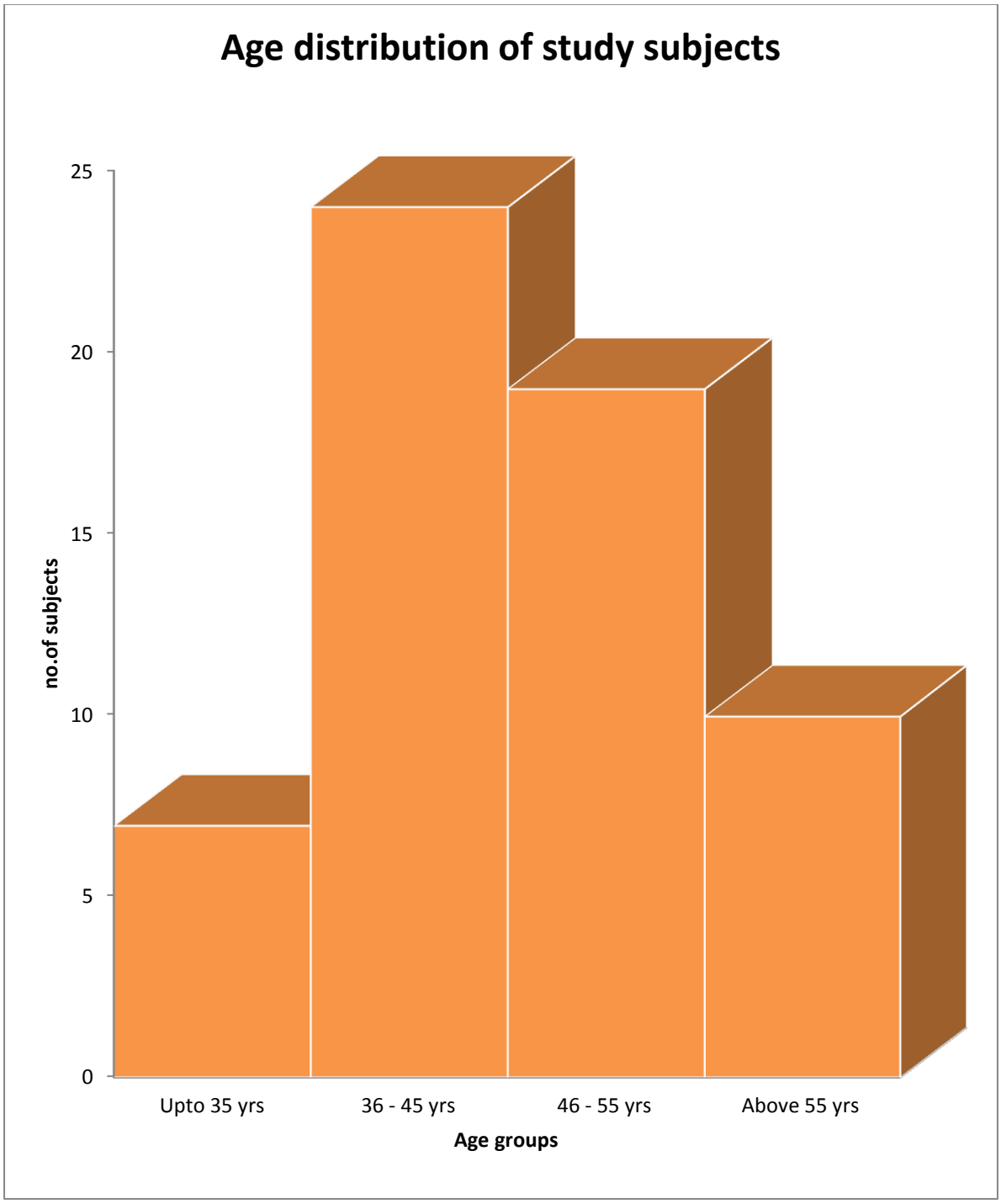


Figure 6: Frequency of MS parameters in study subjects

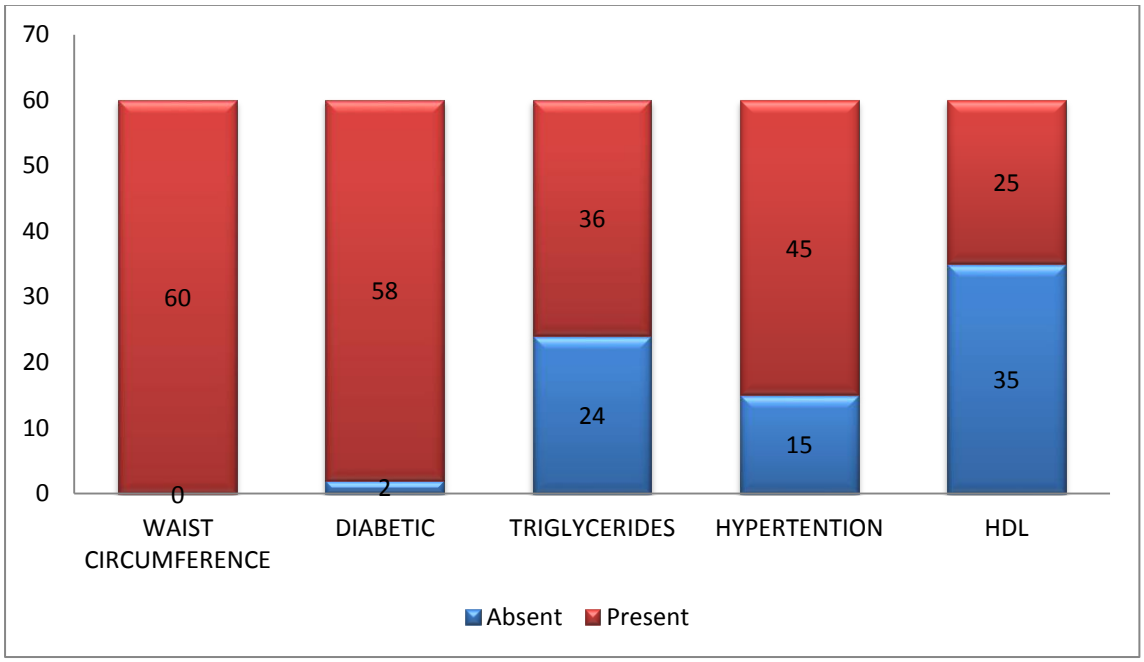


Table 27: Frequency table – MS parameters in study subjects.

	WAIST CIRCUMFERENCE	DIABETIC	TRIGLYCERIDES	HYPERTENTION	HDL
Absent	0	2	24	15	35
Present	60	58	36	45	25

Figure 7: Thyroid status of study subjects

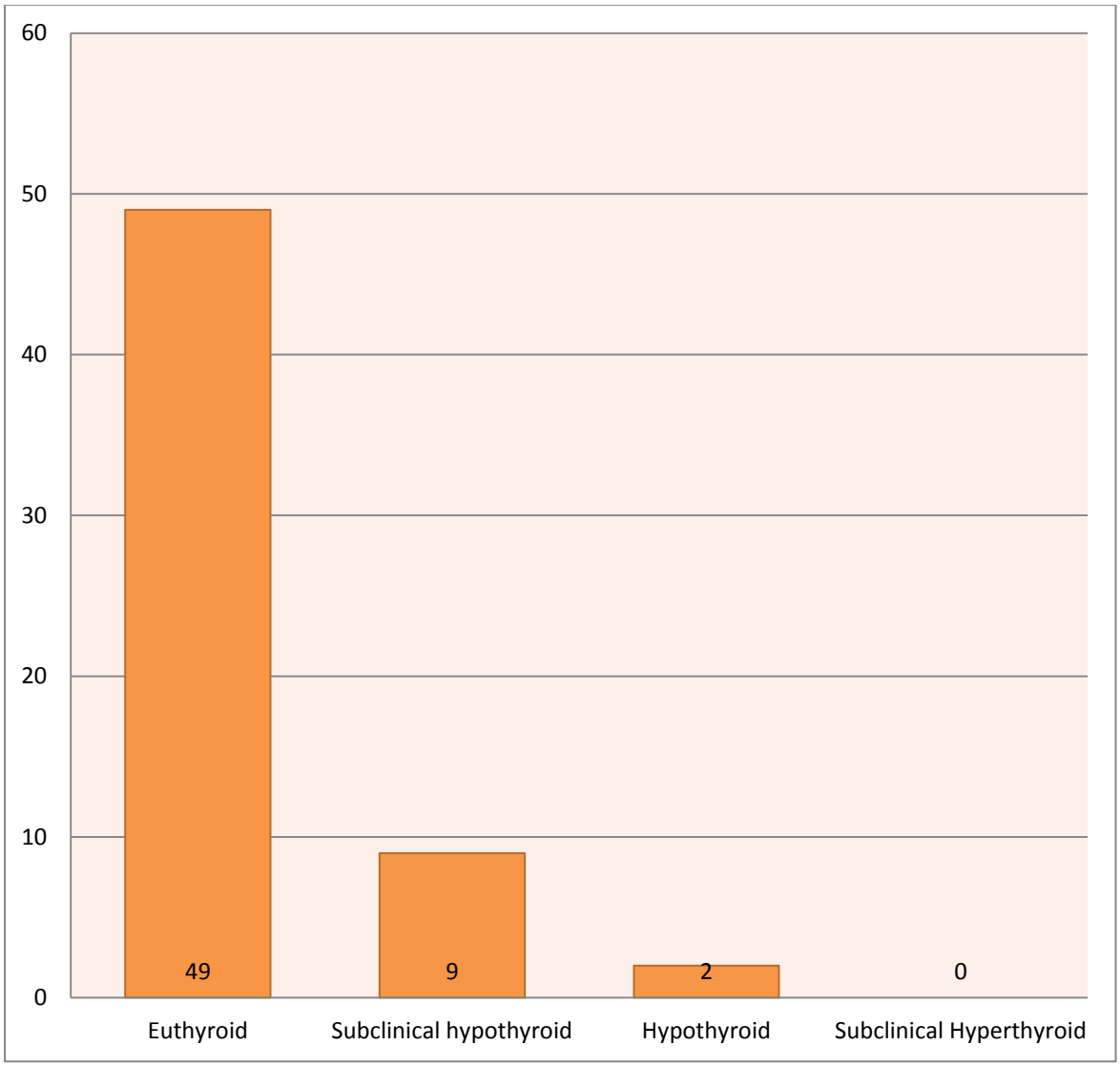


Figure 8: MS parameter – Sex Distribution

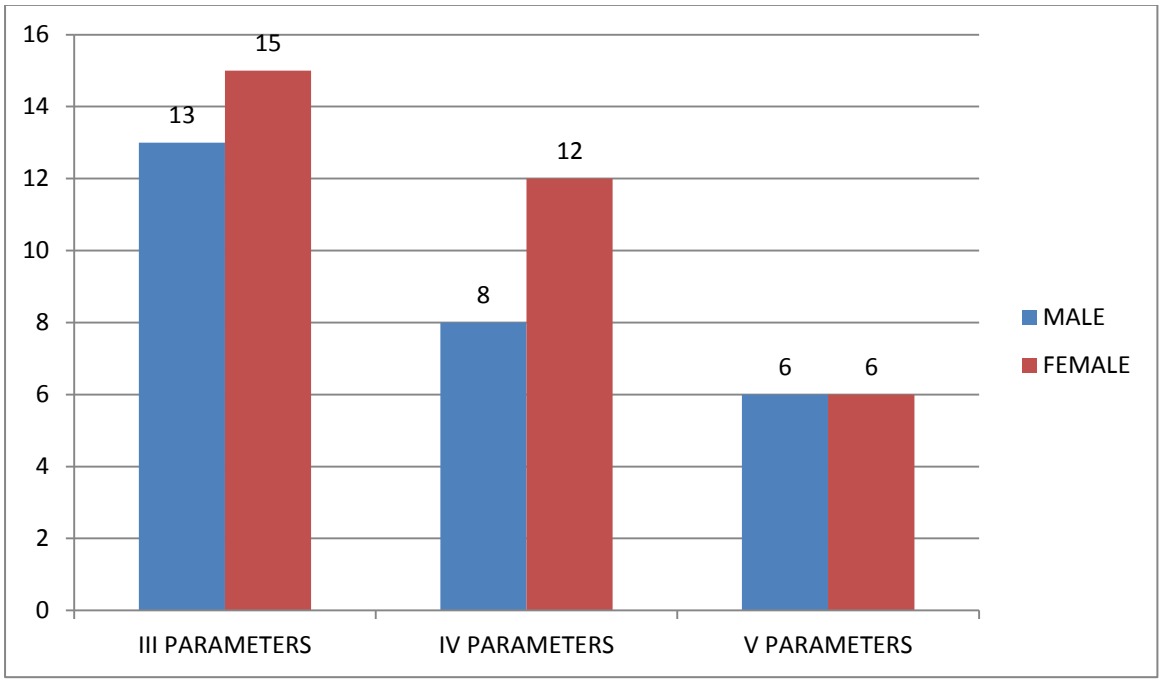


Figure 9: Thyroid status – Age Distribution

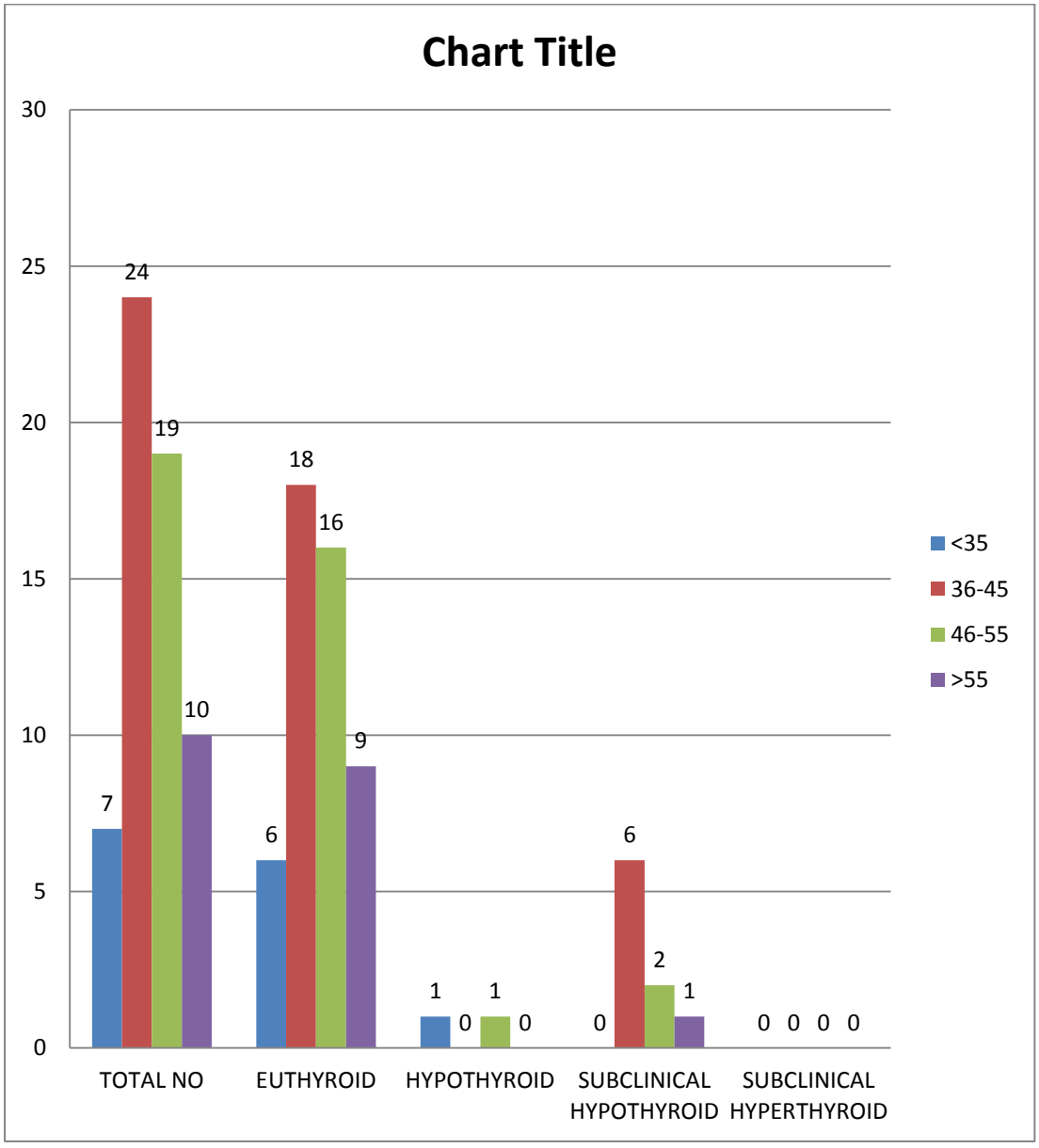


Figure 10: Thyroid status vs. MS parameter

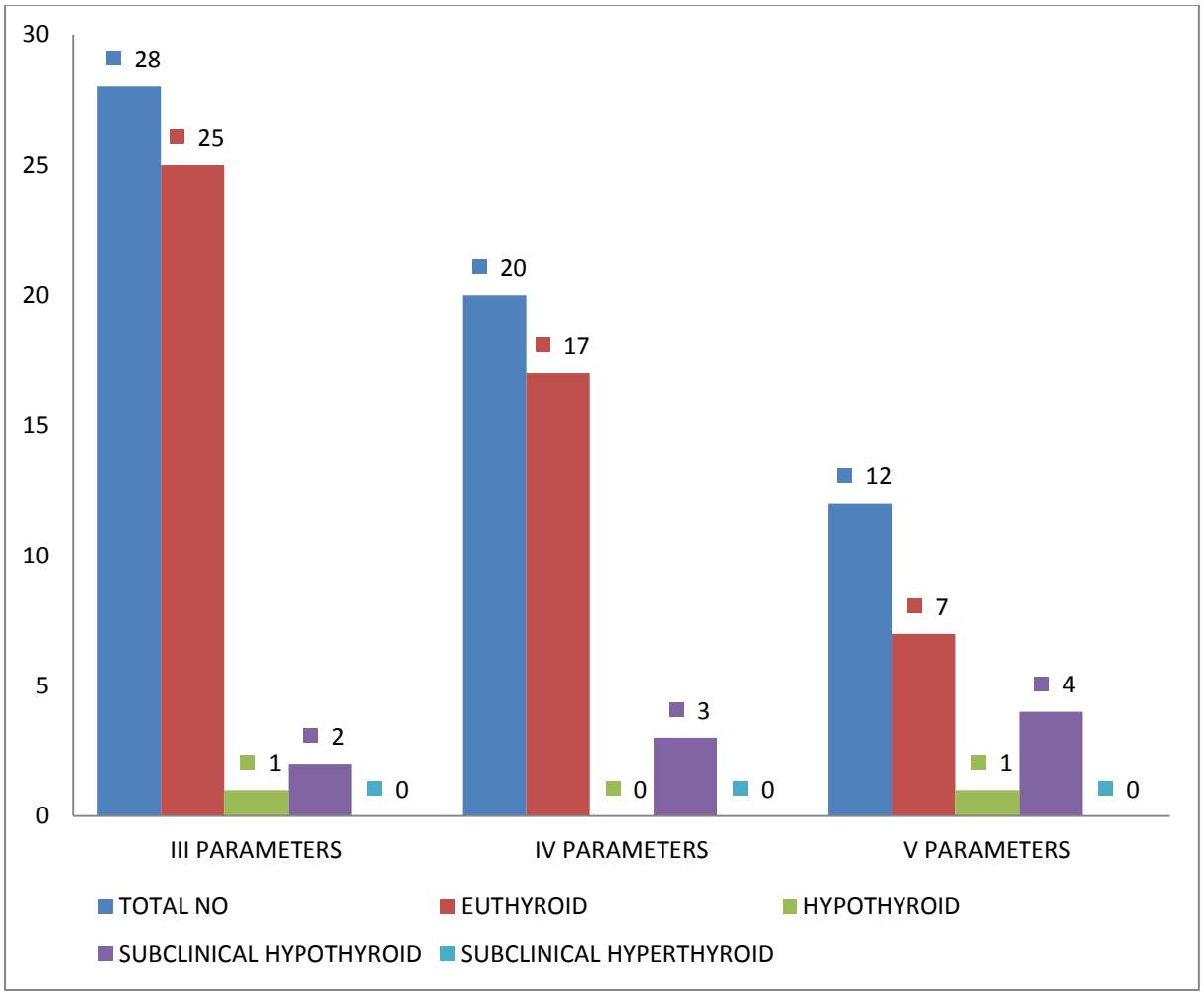


Figure 11: Thyroid status sex distribution

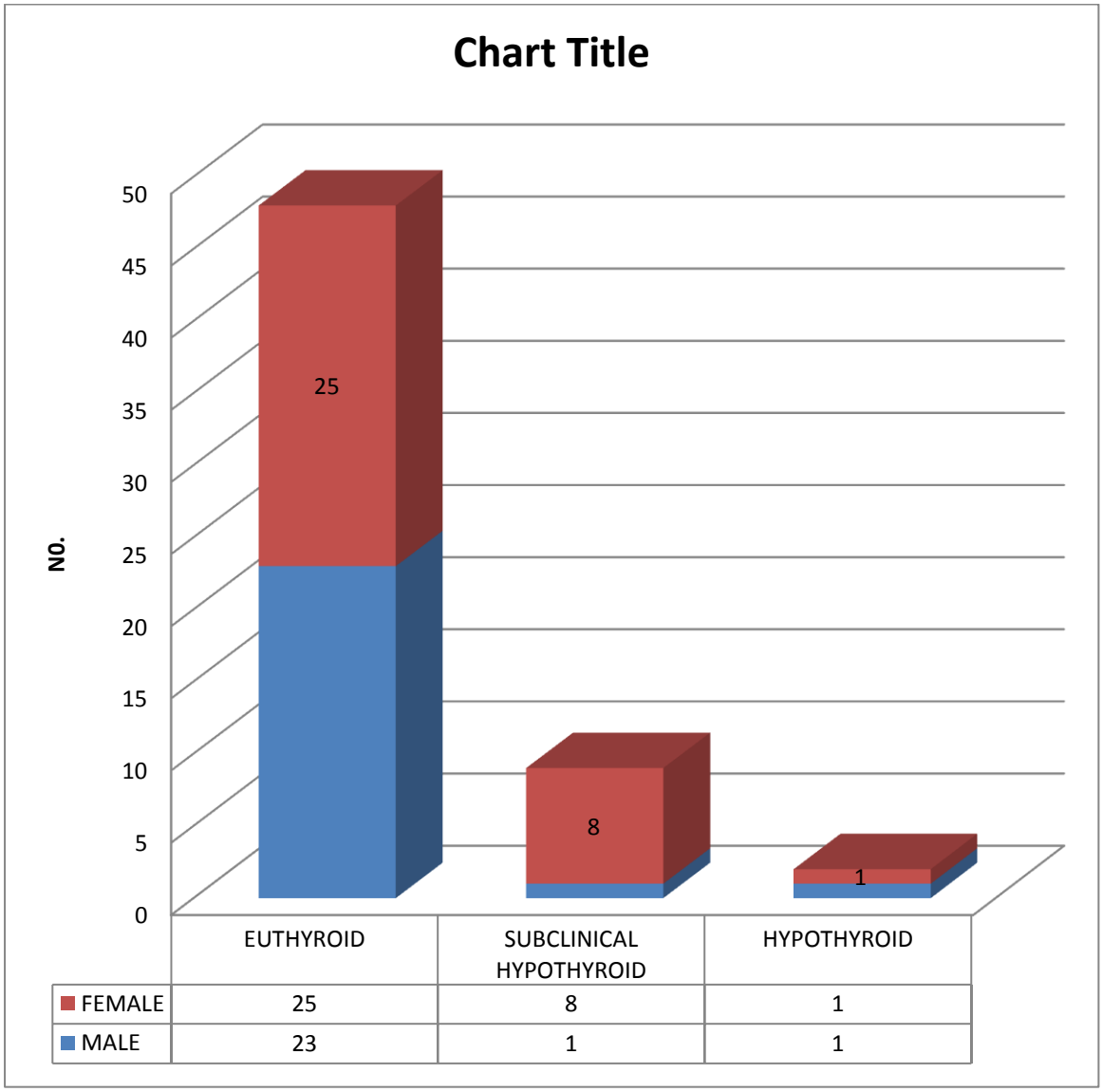
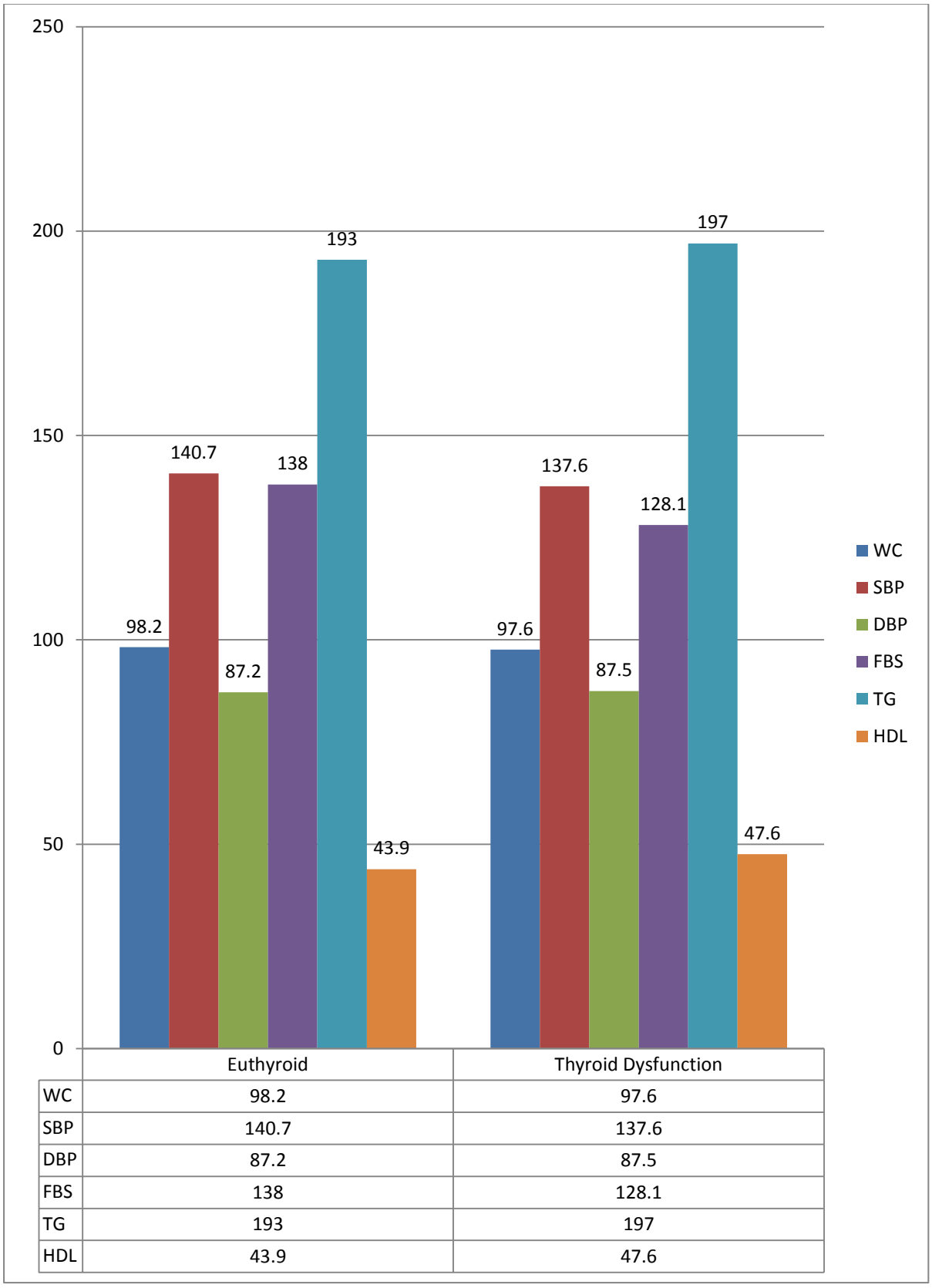


Figure 12: Mean MS parameter in Euthyroid and Thyroid Dysfunction



	Euthyroid	Thyroid Dysfunction
WC	98.2	97.6
SBP	140.7	137.6
DBP	87.2	87.5
FBS	138	128.1
TG	193	197
HDL	43.9	47.6

Discussion

DISCUSSION

"The metabolic syndrome is a cluster of metabolic abnormalities wherein people are obese and have hypertension, high triglyceride level, low high density lipoprotein cholesterol and abnormal fasting glucose levels. (4) People with metabolic syndrome are at high risk for developing cardiovascular disease and type-2 diabetes." Hypothyroidism is associated with lipid abnormalities like high triglycerides and low high density lipoproteins, weight gain, glucose intolerance and hypertension. (32) Thus hypothyroidism mimics the parameters of metabolic syndrome.

In this study, thyroid dysfunction prevalence is 18.33% among metabolic syndrome patients. Subclinical Hypothyroidism is 15% prevalent in metabolic syndrome patients and Overt Hypothyroidism is 3.3% prevalent. There is no incidence of either overt or subclinical Hyperthyroidism in our study population. The prevalence of thyroid dysfunction and hypothyroidism in metabolic syndrome patients are higher than the prevalence in normal population, which is 5.9% for thyroid dysfunction and 4.6% for hypothyroidism (0.3% overt and 4.3% sub clinical hypothyroidism)(34). This study is consistent with study done by Uzunulu et al, as 16.4% of metabolic syndrome patients had hypothyroidism in (35, 44, 46) Japan. (10) In this study prevalence of subclinical hyperthyroidism is 1.7% and there is no overt or clinical hyperthyroidism.

In this study one sixth of metabolic syndrome patients or every sixth

patient with metabolic syndrome has Subclinical hypothyroidism. And one in every 30 patients has overt hypothyroidism. In these hypothyroidism patients, treatment with levothyroxine replacement reverses the symptoms and signs of hypothyroidism, thereby those factors which mimic metabolic syndrome.

It is well known and proven that, by treating with levothyroxine replacement in all overt or clinical hypothyroid patients, we can reduce all the metabolic parameters and cardiovascular risk. (32) Controversy in treating sub-clinical hypothyroidism patients.

Managements of patients sub clinical hypothyroidism remain controversial because the body of scientific evidence available to guide clinical decision is limited. The risk of progression from subclinical hypothyroidism to overt hypothyroid is 2-5% per year. (46) A meta-analysis report shows that levothyroxine therapy in individuals with sub clinical hypothyroidism lowers mean serum total and low density cholesterol concentration significantly and the reduction in serum cholesterol may be larger in individuals with higher pre-treatment cholesterol levels. (47) Another double blind placebo-controlled trial (Basal Thyroid Study) shows that an important risk reduction of cardiovascular mortality of 9 – 31% possible by improvement in low density lipoprotein cholesterol in sub clinical hypothyroidism patients treated with levothyroxine therapy. (48, 51) Surks et al., recommends treating sub clinical hypothyroidism associated with type 2 diabetes and hypertension in his scientific review. (46) As the metabolic syndrome patients have hyperlipidaemia, diabetes, hypertension and increased cardiovascular risk, its look logical to treat

metabolic syndrome patients having sub clinical hypothyroidism by levothyroxine replacement therapy. While there appears to be no adverse effects of initiating levothyroxine treatment in this setting, inadvertent overtreatment occurs in 14-21% of levothyroxine treated patients,(49, 50) carrying potential risks of osteoporosis and atrial fibrillation when serum TSH falls below 0.1 mU/L.(52) These patient need frequent thyroid function tests to avoid this complication.

This study shows that the prevalence of thyroid dysfunction in metabolic syndrome patients is higher than in normal subjects. One sixth of metabolic syndrome patients or every sixth metabolic syndrome had hypothyroidism either overt or subclinical. This finding indicates a need for investigating the presence of Thyroid dysfunction during managing metabolic syndrome patients. As shown in previous evidences, managing these hypothyroid in metabolic syndrome patients are rewarding by improvement in the metabolic parameters and reducing cardiovascular risk.

Conclusions

CONCLUSIONS

1. Thyroid dysfunction occurs in metabolic syndrome patients.
2. Thyroid dysfunction occurs in 18.33% of metabolic syndrome patients.
3. Prevalence of Subclinical hypothyroidism is 15.0% in metabolic syndrome patients which is higher than that of general population.
4. Prevalence of Overt Hypothyroidism is 3.33% in metabolic syndrome patients which is higher than that of general population.
5. One sixth of metabolic syndrome patients or every sixth metabolic syndrome had Subclinical Hypothyroidism.
6. One in every thirty metabolic syndrome patients had Subclinical Hypothyroidism.
7. Prevalence of thyroid dysfunction is much more common in Females with thyroid dysfunction than male.
8. Exclude the presence of Thyroid dysfunction while managing metabolic syndrome patients.

Summary

SUMMARY

Patients with metabolic syndrome have many symptoms and signs suggestive of thyroid dysfunction. It is very difficult to exclude the diagnosis of hypothyroidism on clinical grounds. So a study was conducted in metabolic syndrome patients to study the prevalence and types of thyroid dysfunction.

Based on IDF (2005) criteria for Metabolic Syndrome, Sixty newly detected metabolic syndrome patients attending the outpatient department in Government Kilpauk Medical College and Hospital, Chennai were studied, after taking consent. A detailed history and clinical examination were done. Investigations including metabolic syndrome criteria parameters and serum free T4, TSH were done.

According to thyroid function test results, 49 patients found to have euthyroid and two patients were hypothyroid. Nine patients had sub clinical hypothyroidism. No one in our study had either sub clinical or overt hyperthyroidism.

The thyroid dysfunction is 18.33% prevalent in metabolic syndrome patients. Among the thyroid dysfunction, sub clinical hypothyroidism is highly prevalent – 15.0%. The overt hypothyroidism is 3.3% prevalent in metabolic syndrome patients .

Thyroid dysfunction is much more prevalent in women with metabolic syndrome than men. Nine (40%) out of 35 women had thyroid dysfunction(1 hypothyroid and 8 subclinical hypothyroid). Two (8%) out of 25 men had

thyroid dysfunction (one hypothyroid and one subclinical hypothyroid).

In this study clearly shows that the prevalence of thyroid dysfunction in metabolic syndrome patients is higher than normal subjects. One sixth of metabolic syndrome patients or every sixth metabolic syndrome had Subclinical Hypothyroidism. One in every thirty metabolic syndrome patients had Subclinical Hypothyroidism This finding indicates a need for investigating the presence of Thyroid dysfunction during managing metabolic syndrome patients.

BIBLIOGRAPHY

1. Reaven GM., Banting lecture 1998. Role of insulin resistance in human disease. Diabetes 1988; 37: 1595-1607.
2. Reaven GM et al., Insulin resistance, cardiovascular disease and the metabolic syndrome. Diabetes Care 2004; 27: 1011-12.
3. Grundy SM et al., Obesity, metabolic syndrome and cardio vascular disease. J Clin Endocrinol Metab 2004;89: 2595-2600.
4. Zimmet PZ et al ., The metabolic syndrome. : Lancet 2005; 365: 1415-1428.
5. Cappola AR et al., Hypothyroidism and atherosclerosis J Clin Endocrinol Metab 2003; 88: 2438-2444.
6. Hak AE et al., Sub clinical hypothyroidism is an independent risk factor foe atherosclerosis and myocardial infarction; The Rotterdam study. Ann Intern Med 2000; 132: 270-278.
7. Klein I et al., Thyroid hormone and cardiovascular system. N Engl J Med 1976; 344: 501-509.
8. Duntas LH., Thyroid disease and lipids. Thyroid 2002; 12:287-293.
9. Dillman WH et al., Mechanism of action of thyroid hormones. Med Clin North Am 1985;69: 849
10. Uzunulu M et al., Prevalence of sub clinical hypothyroidism in patients with metabolic syndrome. Endocr J. 2007; 54: 171 - 76.
11. Chandra L et al., Association of metabolic syndrome and its components

with thyroid dysfunction in females. Int J Diab Dev Ctries 2007; 27:124-26.

12. Defranzo RA et al., A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia and atherosclerotic cardiovascular disease. Diabetes Care 1991; 14:173-94.
13. Kaplan NM et al., 1989 The deadly quartet. Arch Intern Med 1989; 149: 1514-20.
14. Isomaa B et al., Cardiovascular morbidity and mortality associated with metabolic syndrome. Diabetes Care 2001; 24: 683-89.
15. Lakka HM et al., The metabolic syndrome and cardiovascular disease mortality. JAMA 2002; 228: 2709-16.
16. Kyelin E et al., Hypertonie- hyperglykamie- hyperurikaemic syndrome. Zentralblatt fur innere Medizine 1923 (44).
17. Alberti KG et al., Diagnosis and definition - provisional report of WHO consultation. Diabet Med 1998; 15: 539-553.
18. National Cholesterol Education Programme: Adult Treatment Panel III (NCEP: ATP III)., Expert panel on detection, evaluation and treatment of high blood cholesterol in adults. JAMA 2001; 285: 2486-2497.
19. Balkau B et al., European Group for study of Insulin Resistance (EGIR). Diabet Med 1999; 16: 442-443.
20. WHO., Definition, diagnosis and classification of Diabetes Mellitus. (1999).
21. AHA/NHLBI Scientific Statement., Diagnosis and Management of the

metabolic syndrome. Circulation 2005;112:285-290.

22. The IDF consensus - May 2004 Worldwide definition of the metabolic syndrome. www.idf.org

23. Tan CE et al., Can we apply NCEP-ATP III definition for the metabolic syndrome to Asians? Diabetes Care 2004; 27: 1182-6.

24. WHO, Western Pacific Region., Redefining obesity and its treatment. WHO/IASO/IOTF,2000.

25. Ford ES et al., The prevalence of metabolic syndrome among US adults. JAMA 2002; 287: 356-59.

26. Mohan V et al., The prevalence of metabolic syndrome in a selected south Indian population. Indian J Med Res 2002; 115: 118-27.

27. Ramachandran A et al., Metabolic syndrome in urban Asian Indian Adults. Diab Res Clin Pract 2003; 60: 199-204.

28. Boden G et al., Free fatty acids in obesity and type 2 diabetes – Insulin resistance. Eur J Clin Invest 2002; 32(suppl 3): 14-23.

29. Jean PD et al., RIO-Lipids study group. Effects of rimonabant on metabolic risk factors in overweight patients with dyslipidemia. N Eng J Med 2005; 353: 2121-34.

30. Wierzbicki et al., Rimonabant: endocannabinoid inhibition for the metabolic syndrome. Int J Clin Pract 2006;60:12:1697-1706.

31. Degroot, The thyroid and its diseases, 6th Edition.

32. Williams., Endocrinology 10th Edition

33. Gay J C et al., The Colorado thyroid disease prevalence study. Arch Intern

Med 2000; 160: 526-534.

34. NHANES III., Serum TSH, T4, and thyroid antibodies in the United States population. J Clin Endocrinol Metab 2002; 87(2): 489-499.

35. Consensus statement: Sub clinical thyroid dysfunction: A joint statement from the American Association of Clinical Endocrinologists, the American Thyroid Association, and The Endocrine Society. J Clin Endocrinol Metab 2005; 90(1):581-585.

36. Cooper DS et al., Sub clinical Hupothyroidism. N Eng J Med 2001; 345: 260-265.

37. Zulewski H et al., Evaluation of patients with various grades of hypothyroidism. J Clin Endocrinol Metab 1997;82:771-776.

38. Klein I et al., Thyroid hormone and the cardio vascular system. N Eng J Med 2001; 344: 501-509.

39. Polikar R et al., The thyroid and the heart. Circulation 1993; 87: 1435-1441.

40. Daese MD et al., Effect of thyroxin therapy on serum lipoproteins in patients with mild thyroid failure. J Clin Endocrinol Metab 2000; 22: 153-158.

41. Dullaart RPF et al., The activity of cholesterol ester transfer protein is decreased in hypothyriodism. Eur J Clin Invest 1990; 20: 581-587.

42. Annemieke R et al., Thyroid function is associated with components of the metabolic syndrome in euthyroid subjects. J Clin Endocrinol Metab 2007; 92(2): 491-496.

43. Stephan J et al., The relationship between thyrotropin and lipoprotein cholesterol is modified by insulin sensitivity in healthy euthyroid subjects. J Clin Endocrinol Metab 2001; 86: 1206-1211.

44. Marina AM et al., Thyroid function in humans with morbid obesity. Thyroid (Ind Ed) 2006; 1(2): 37-42.

45. Surks MI et al., Subclinical Thyroid Disease: Guidelines for diagnosis and management. JAMA 2004; 291: 228-238.

46. Surks MI et al., Subclinical Thyroid Disease: Clinical applications. JAMA 2004; 291: 239-243.

47. Mark D et al., Effect of thyroxine therapy on lipoproteins in patients with mild thyroid failure. J Clin Endocrinol Metab 2000; 85: 2993-3001.

48. Meier C et al., TSH controlled L-thyroxine therapy reduces cholesterol level and clinical symptoms in sub clinical hypothyroidism. J Clin Endocrinol Metab 2001; 86: 4860-4866.

49. Ross DS et al., The use and limitation of thyrotropin assay as a single thyroid function test. J Clin Endocrinol Metab 1990; 71: 764-769.

50. Parle JV et al., Thyroxine prescription in the community. Br J Gen Pract 1993; 43: 107-109.

51. Nadia C et al., Lipoprotein profile in Subclinical hypothyroidism: response to levothyroxine therapy. J Clin Endocrinol Metab 2002; 87: 1533-1538.

52. Cappola AR et al., Thyroid status, cardiovascular risk and mortality in older adults. JAMA 2006; 295: 1033-1041.

LIST OF TABLES

LIST OF FIGURES

PROFORMA

PERSONAL DETAILS

NAME
AGE
SEX
ADDRESS

PHONE NO.
EDUCATION
OCCUPATION

TYPE — PROFESSIONAL
SEMI SKILLED
UNSKILLED(MANUAL WORKER)

MONTHLY INCOME

EXCLUSION CRITERIA:

a. Known Hypothyroid / Subclinical Hypothyroid
b. Patients with chronic illness
c. Taking Steroids
d. Severely ill patients
e. Pregnant Women
f. Individuals below 18 Yrs.

MEASUREMENTS

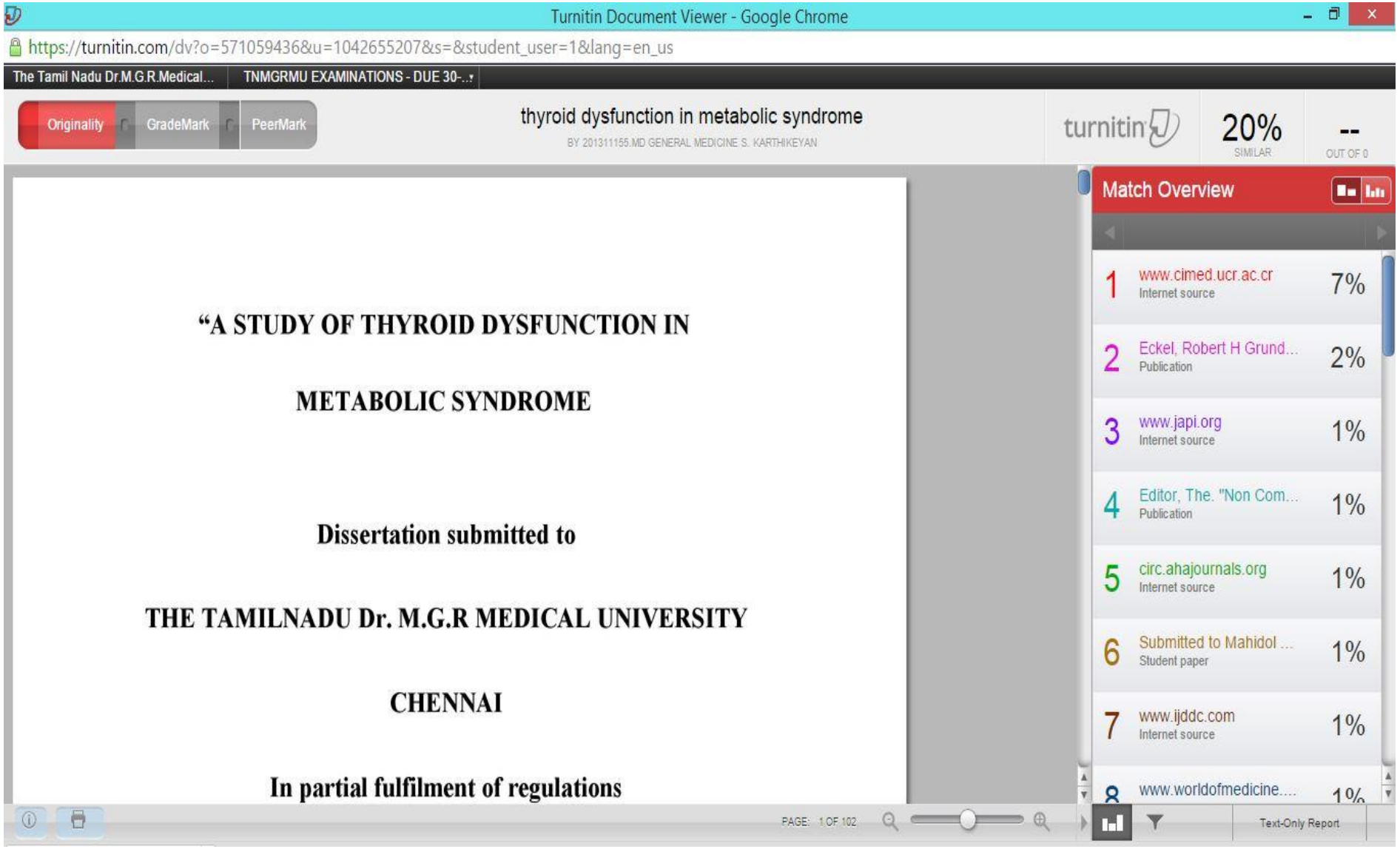
Height : cm
Weight Kg
BMI : Kg/M^2
Waist Circumference : cm

VITALS

Heart Rate :		beats / mt
Blood Pressure :	Systolic :	mm of Hg
	Diastolic :	mm of Hg

BIOCHEMISTRY

Fasting Blood Sugar		mgs%
	Lipid Profile	
		mgs%
Total Cholesterol		mmol%
HDL – C		mgs% mmol%
TG		mgs% mmol%
	Thyroid profile	
TSH		mU/L
FT4		ng/dl


Turnitin Document Viewer - Google Chrome
https://turnitin.com/dv?o=571059436&u=1042655207&s=&student_user=1&lang=en_us
The Tamil Nadu Dr.M.G.R.Medical...
TNMGRMU EXAMINATIONS - DUE 30-..
Originality
GradeMark
PeerMark
thyroid dysfunction in metabolic syndrome
BY 201311155.MD GENERAL MEDICINE S. KARTHIKEYAN
turnitin
20%
SIMILAR
--
OUT OF 0
"A STUDY OF THYROID DYSFUNCTION IN
METABOLIC SYNDROME
Dissertation submitted to
THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY
CHENNAI
In partial fulfilment of regulations
Match Overview
1 www.cimed.ucr.ac.cr Internet source 7%
2 Eckel, Robert H Grund... Publication 2%
3 www.japi.org Internet source 1%
4 Editor, The. "Non Com... Publication 1%
5 circ.ahajournals.org Internet source 1%
6 Submitted to Mahidol ... Student paper 1%
7 www.ijddc.com Internet source 1%
8 www.worldofmedicine.... 1%
PAGE: 1 OF 102
Text-Only Report

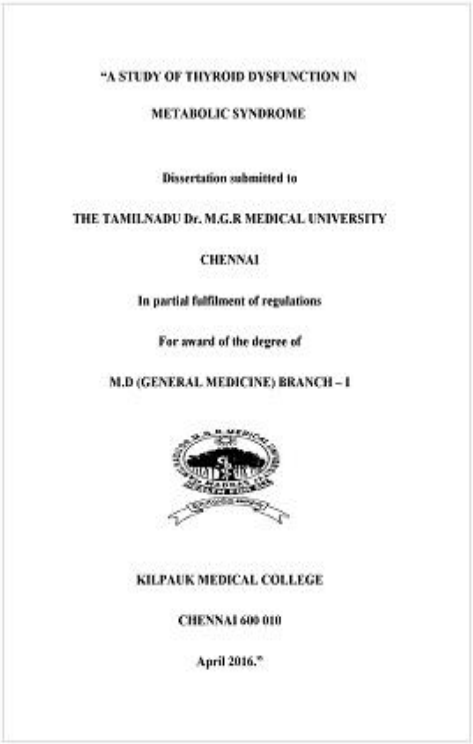
turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	201311155.md General Medicine S....
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	thyroid dysfunction in metabolic sy...
File name:	thyroid_dysfunction_in_metabolic_...
File size:	39.11K
Page count:	102
Word count:	11,251
Character count:	60,884
Submission date:	06-Oct-2015 03:30AM
Submission ID:	571059436



"A STUDY OF THYROID DYSFUNCTION IN

METABOLIC SYNDROME

Dissertation submitted to

THE TAMILNADU Dr. M.G.R MEDICAL UNIVERSITY

CHENNAI

In partial fulfilment of regulations

For award of the degree of

M.D (GENERAL MEDICINE) BRANCH – I

KILPAUK MEDICAL COLLEGE

CHENNAI 600 010

April 2016."

MASTER CHART

MASTER CHART PAGE 1

NO	NAME	AGE	SEX	HEIGHT	WEIGHT	BMI	WC	SBP	DBP	FBS	TC	HDL	TG	FT4	TSH	TSH_G	MS _P
1	SATHYA.H	49	2	146	82	28.0822	87	132	86	118	164	48	82	1.39	1.14	2	3
2	REVATHI.P	36	2	149	80	26.8456	107	142	90	103	161	48	160	1.69	1.82	2	4
3	SARAVANAN.	30	1	168	86	25.5952	94	124	84	130	224	44	109	0.17	154	4	3
4	PURUSOTHAMAN K	51	1	158	78	24.6835	96	136	78	120	177	44	106	0.97	4.71	3	4
5	VIJAYA.R	33	2	150	88	29.3333	86	128	86	106	212	43	167	0.97	4.1	2	3
6	KARTHIGA.S	43	2	144	69	23.9583	102	148	100	142	154	54	160	0.81	7.78	3	5
7	RAJA LAKSHMI.K	55	2	148	74	25	96	144	90	114	148	49	148	1.1	1.12	2	3
8	SAGUNTHALA	57	2	162	106	32.7161	116	150	92	122	171	38	114	1.03	5.57	3	3
9	BABU YUVARAJ.B	47	1	171	96	28.0702	100	124	72	131	189	43	148	1.28	1.88	2	3
10	MEENAKSHI.R	38	2	155	78	25.1613	85	130	96	132	229	52	215	0.95	6.21	3	5
11	KANCHANA.S	52	2	144	78	27.0833	94	156	86	150	134	53	83	1.33	2.74	2	3
12	KARPAKAM.G	58	2	147	78	26.5306	94	140	92	160	199	49	134	1.05	1.56	2	4
13	SRIVIDHYA.V	62	1	168	94	27.9762	102	146	90	146	196	44	242	0.86	3.06	2	5
14	USHA.D	48	2	157	84	26.7516	90	134	86	116	169	45	162	0.99	7.08	3	4
15	PADMA.K	40	2	149	75	25.1678	85	108	66	144	213	52	155	0.82	2.45	2	4
16	RAJA.A	62	1	170	96	28.2353	107	170	88	112	273	38	139	1	2.41	2	3
17	BARANI.K	43	1	162	90	27.7778	99	134	82	108	178	45	107	0.95	1.56	2	3
18	SARANYA.R	39	2	157	83	26.4331	98	126	72	122	167	50	174	0.92	2.02	2	4
19	RAJASEKAR.L	67	1	166	85	25.6024	94	134	86	142	152	38	87	1.44	0.91	2	3
20	LAKSHMI.G	46	2	148	72	24.3243	88	126	76	104	179	52	168	1.02	1.19	2	4

TSH_G

1 - SUBCLINICAL HYPERTYHYROID

2 - EUTHYROID

3 - SUBCLINICAL HYPOTHYROID

4 – HYPOTHYROID

MASTER CHART PAGE 2

NO	NAME	AGE	SEX	HEIGHT	WEIGHT	BMI	WC	SBP	DBP	FBS	TC	HDL	TG	FT4	TSH	TSH_G	MS _P
21	SARALA.P	37	2	163	87	26.68712	108	134	90	156	199	52	214	1.31	1.01	2	5
22	JUNAITHA.B	49	2	152	76	25	102	156	100	96	187	51	199	1.28	3.52	2	4
23	JHONSON.M	61	1	165	89	26.9697	97	142	88	112	242	42	196	1.31	1.19	2	5
24	JANAKI.D	36	2	147	77	26.19048	94	140	80	152	148	48	96	1.01	1.55	2	3
25	ROOPA.J	37	2	157	82	26.11465	102	144	90	148	147	48	77	1.39	1.4	2	3
26	SHANTHI.M	40	2	149	92	30.87248	108	100	70	110	200	44	127	0.78	9.98	3	3
27	SELVI.R	45	2	160	100	31.25	106	102	66	112	175	47	225	1.05	0.56	2	3
28	JANSI.I	39	2	146	76	26.0274	94	146	86	126	234	51	224	0.9	5.28	3	5
29	RAMKUMAR.J	42	1	157	86	27.38854	101	110	70	114	217	44	212	0.99	1.83	2	4
30	SHANMUGAM.K	57	1	180	106	29.44444	106	172	90	128	216	40	178	0.96	3.39	2	5
31	RAJESWARI.T	50	2	159	82	25.78616	92	144	86	123	210	52	196	0.68	24.24	4	5
32	ARASI.P	50	2	152	78	25.65789	92	146	90	116	216	45	166	1.24	1.54	2	3
33	MOHAN.N	35	1	166	88	26.50602	95	152	100	120	192	45	175	0.95	2.07	2	5
34	ARAVINDAN.K	48	1	170	98	28.82353	102	120	80	132	156	49	145	1.14	1.9	2	3
35	JANAKI.P	47	2	148	76	25.67568	92	144	96	166	231	43	177	0.96	1.96	2	4
36	PINKY.K	40	2	156	83	26.60256	100	142	84	122	193	56	172	0.85	6.42	3	5
37	MUNIRATHINAM.P	47	1	152	85	27.96053	96	150	90	136	173	54	129	0.96	2.24	2	3
38	BAKYARAJ.A	42	1	169	93	27.51479	96	152	90	162	291	43	182	1.12	1.71	2	5
39	SELVAKUMAR.B	45	1	149	83	27.85235	86	136	90	105	174	49	149	0.99	0.99	2	3
40	DURAI.J	45	1	165	82	24.84848	92	146	92	124	193	39	122	0.95	1.17	2	3

TSH_G

1 - SUBCLINICAL HYPERTYHYROID

2 - EUTHYROID

3 - SUBCLINICAL HYPOTHYROID

4 - HYPOTHYROID

MASTER CHART PAGE 3

NO	NAME	AGE	SEX	HEIGHT	WEIGHT	BMI	WC	SBP	DBP	FBS	TC	HDL	TG	FT4	TSH	TSH_G	MS _P
41	RATHINAMALA.B	62	2	161	87	27.01863	106	130	90	162	191	47	116	0.93	1.13	2	3
42	SADAGOPAN.S	46	1	172	90	26.16279	97	140	90	156	199	42	297	2.21	1.12	2	5
43	RAGUVARAN.M	48	1	161	89	27.63975	116	136	90	184	201	45	146	1.16	3.54	2	4
44	SARAVANAN.I	40	1	160	96	30	95	152	94	128	255	38	180	1.13	0.88	2	4
45	BANU.F	40	2	164	92	28.04878	92	138	82	144	234	32	198	1.08	1.55	2	4
46	MURUGAN.A	38	1	167	87	26.0479	99	126	70	154	149	31	206	0.92	1.83	2	3
47	SHIEK.D	48	1	159	77	24.21384	94	134	82	166	208	49	125	1.14	1.42	2	3
48	IQBAL.R	45	1	164	94	28.65854	98	170	110	189	260	31	296	1.11	1.11	2	3
49	MADHAVI.G	53	2	154	79	25.64935	109	168	106	152	183	49	144	1.24	0.92	2	3
50	RANJITH	60	1	152	80	26.31579	95	146	70	200	191	31	231	1.11	0.57	2	4
51	JAMUNA.B	44	2	148	76	25.67568	100	126	70	122	153	47	242	0.96	1.4	2	3
52	JAMES.K	35	1	160	91	28.4375	107	172	106	166	207	38	222	1.06	0.91	2	4
53	KAMAKSHI.T	32	1	172	88	25.5814	104	136	92	104	174	37	214	0.98	1.58	2	4
54	SATHYA.L	43	2	151	80	26.49007	93	150	100	140	204	35	184	0.99	2.76	2	4
55	MANORAMMA.G	52	2	153	92	30.06536	96	130	80	170	155	49	174	0.82	1.58	2	3
56	MAHESWARI.M	35	1	174	88	25.28736	104	140	86	118	193	45	148	1.17	2.04	2	3
57	SEETHA.R	42	2	142	80	28.16901	97	160	100	144	210	40	192	0.87	6.38	3	4
58	RAJALAKSHMI.S	56	2	159	90	28.30189	99	148	96	156	228	38	114	1.17	1.5	2	3
59	HARIKRISHNAN.J	47	1	161	91	28.26087	97	134	90	142	198	31	307	1.29	2.05	2	4
60	DEEPA.S	35	2	147	74	25.17007	106	160	104	172	165	46	236	1.33	1.92	2	4

TSH_G

1 - SUBCLINICAL HYPERTYHYROID

2 - EUTHYROID

3 - SUBCLINICAL HYPOTHYROID

4 - HYPOTHYROID